Essential Thrombocytosis as a Service-Related Condition in Canadian Armed Forces Combat Engineers: An Evidence-Based Report for Veterans Affairs Canada

Section 1: Executive Summary

Purpose and Scope

This report presents a comprehensive, evidence-based argument for Veterans Affairs Canada (VAC) to recognize Essential Thrombocytosis (ET), a myeloproliferative neoplasm (MPN), as a service-related condition for Canadian Armed Forces (CAF) Combat Engineers. The analysis synthesizes current medical-scientific understanding of ET's pathophysiology, the specific occupational hazards inherent to the Combat Engineer trade, and the toxicological and epidemiological evidence linking these exposures to the disease's molecular origins. The scope of this report encompasses the full spectrum of a Combat Engineer's career, including training, domestic operations, and international deployments, with a particular focus on the complex exposure environment of the Afghanistan theatre of operations.

Core Argument

The central thesis of this report is founded on three logically interconnected pillars of evidence, which collectively establish a compelling case for service connection:

1. **The Nature of the Disease:** Essential Thrombocytosis is a clonal blood cancer initiated by *acquired* somatic mutations in hematopoietic (blood-forming) stem cells. The disease is not primarily an inherited condition but one that develops during an individual's lifetime due to genetic damage. The vast majority of cases are driven by specific, identifiable mutations in the *Janus kinase 2* (*JAK2*), *calreticulin* (*CALR*), or *myeloproliferative leukemia virus oncogene* (*MPL*) genes, which cause the uncontrolled production of platelets. The acquired nature of these mutations creates a biological vulnerability to external, genotoxic agents.

2. **The Nature of the Occupation:** The duties of a CAF Combat Engineer entail chronic, repeated, and multifaceted exposure to a synergistic cocktail of known and suspected human carcinogens, hematotoxins (agents toxic to blood and bone marrow), and genotoxins (agents that damage DNA). These hazardous exposures are not incidental but are integral to the core functions of the trade. They include, but are not limited to, benzene and other volatile organic compounds from fuels and solvents; polycyclic aromatic hydrocarbons (PAHs) from diesel exhaust; the chemical constituents and

detonation byproducts of military-grade explosives such as RDX and TNT; and a complex mixture of toxicants from open-air burn pits, including dioxins, furans, and heavy metals.

3. **The Causal Link:** A scientifically plausible and compelling mechanistic pathway exists wherein the service-related chemical exposures encountered by Combat Engineers can induce the specific types of DNA damage that result in the hallmark genetic mutations of ET. Exposure to benzene and PAHs, in particular, is known to target hematopoietic stem cells, promote a pre-cancerous state known as clonal hematopoiesis, and is epidemiologically linked to other *JAK2*-driven myeloproliferative neoplasms.

Key Evidence

The case presented herein is substantiated by a robust body of evidence drawn from peer-reviewed medical literature, government toxicological reports, and epidemiological studies of veteran populations. Toxicological data confirm the hematotoxic and carcinogenic properties of chemicals central to a Combat Engineer's work environment. Epidemiological research on large cohorts of U.S. veterans demonstrates a significantly elevated incidence of MPNs among those who served in the Persian Gulf War era, including in Afghanistan, with an earlier age of onset and a more aggressive disease course. Furthermore, the policy precedent set by allied nations, notably the United States Department of Veterans Affairs—which recognizes closely related myeloid neoplasms like leukemias and myelodysplastic syndromes as presumptive conditions for burn pit exposure—provides a powerful framework for policy alignment.

Recommendation

Based on the overwhelming weight of the assembled scientific and occupational evidence, and in accordance with the guiding principles of Veterans Affairs Canada, including the "Benefit of Doubt" principle, this report formally recommends the inclusion of Essential Thrombocytosis as a recognized service-related condition for CAF Combat Engineers. This recognition should apply to members with documented or reasonably inferred exposure to the hazardous agents detailed in this report during their training and/or on domestic or international deployments. Establishing this connection is a necessary step to ensure that veterans suffering from this chronic and potentially life-threatening cancer receive the care, support, and recognition they have rightfully earned through their service to Canada.

Section 2: Understanding Essential Thrombocytosis (ET) as a Myeloproliferative Neoplasm (MPN)

2.1. Clinical Definition and Pathophysiology of ET

Essential thrombocytosis (ET) is a chronic blood cancer formally classified as a myeloproliferative neoplasm (MPN) by the World Health Organization (WHO). The term "myeloproliferative" refers to the uncontrolled growth (proliferation) of cells originating in the bone marrow (myeloid cells). ET is specifically defined by the excessive production of platelets (thrombocytosis), which are the small blood cells responsible for forming clots to stop bleeding. This overproduction stems from a clonal defect in a single hematopoietic stem cell, meaning

that all the abnormal cells are descendants of one original mutant cell. This leads to a marked increase in the number of platelet-forming cells, known as megakaryocytes, within the bone marrow, resulting in the release of an abnormally high number of platelets into the bloodstream. The clinical consequences of ET are primarily thrombo-hemorrhagic. While platelets are essential for hemostasis, their extreme overabundance in ET can lead to paradoxical and dangerous outcomes. The high platelet count, combined with qualitative defects in platelet function and the activation of other blood cells like leukocytes, creates a prothrombotic or hypercoagulable state. This significantly increases the patient's risk of forming blood clots (thrombosis) in both arteries and veins, which can lead to life-threatening events such as heart attack, stroke, or pulmonary embolism. Less commonly, particularly when platelet counts are extremely high (e.g., > 1,000,000/mcL), patients can experience bleeding complications due to an acquired deficiency of a critical clotting protein called von Willebrand factor.

ET is part of a spectrum of closely related MPNs that share pathogenic mechanisms, particularly Polycythemia Vera (PV), characterized by an overproduction of red blood cells, and Primary Myelofibrosis (PMF), characterized by the progressive scarring of the bone marrow. There is significant phenotypic overlap between these conditions, and ET can evolve over time. A proportion of ET patients will develop bone marrow fibrosis, and the disease can transform into the more aggressive conditions of post-ET myelofibrosis or, in a small number of cases, acute myeloid leukemia (AML).

The diagnosis of ET is often one of exclusion, meaning it is confirmed after ruling out other conditions that can cause a high platelet count. These include other MPNs, myelodysplastic syndromes (MDS), and secondary or "reactive" thrombocytosis, which can be caused by underlying conditions like iron deficiency, inflammation, infection, or cancer. To standardize the diagnosis, the WHO has established a set of rigorous criteria that combine clinical, morphological, and genetic findings.

2.2. The Central Role of Acquired Somatic Mutations

A fundamental aspect of ET's pathophysiology, and one that is central to the argument for service connection, is that the disease is overwhelmingly caused by *acquired* genetic mutations. These are not inherited genetic defects present at birth but rather alterations to the DNA of a single blood-forming cell that occur at some point during an individual's lifetime. This establishes a clear biological premise: the onset of ET is triggered by an event or a series of events that cause DNA damage. Genetic mutations can be caused by mistakes during normal cell division or, critically, by exposure to DNA-damaging agents in the environment, such as hazardous chemicals or radiation.

Scientific breakthroughs since 2005 have identified a core group of "driver mutations" that are responsible for approximately 90% of ET cases. These mutations all converge on the same biological pathway—the JAK-STAT signaling pathway—which acts as a master regulator of blood cell production. The three primary driver mutations are:

- **Janus Kinase 2 (*JAK2*) V617F Mutation:** This is the most prevalent genetic abnormality in ET, identified in approximately 50% to 64% of all patients. The *JAK2* gene provides the instructions for making the JAK2 protein, a critical enzyme that transmits growth signals within hematopoietic stem cells. The specific V617F mutation is a point mutation that

causes the JAK2 protein to become permanently activated, or constantly switched "on". This leads to cytokine-independent growth, meaning the bone marrow stem cells proliferate uncontrollably without needing the normal hormonal signals (like thrombopoietin) that regulate platelet production.

- **Calreticulin (*CALR*) Mutation:** Found in about 23% to 25% of ET patients, the *CALR* mutation is the second most common driver and is typically found in patients who do not have the *JAK2* mutation. The mutated CALR protein has been shown to bind to and activate the thrombopoietin receptor (*MPL*), effectively mimicking the body's natural signal to produce more platelets and thereby driving the disease process.

- **Myeloproliferative Leukemia Virus Oncogene (*MPL*) Mutation:** The least common of the three main drivers, *MPL* mutations are found in approximately 3% to 4% of ET patients. The *MPL* gene codes for the thrombopoietin receptor itself. Mutations in this gene lead to its constitutive, ligand-independent activation, again resulting in a constant "on" signal for platelet production.

A small subset of patients, around 10%, are termed "triple-negative" as they lack any of these three canonical mutations. This indicates that other, less common genetic or epigenetic events can also initiate the disease, though the underlying principle of an acquired defect in a hematopoietic stem cell remains the same. The fact that the vast majority of ET cases can be traced back to specific, acquired DNA mutations provides a clear molecular target for potential environmental and occupational mutagens.

2.3. The Molecular Basis of Myeloproliferation: The JAK-STAT Pathway

The JAK-STAT (Janus kinase/signal transducer and activator of transcription) pathway is a crucial intracellular signaling cascade that translates external chemical signals into a cellular response. In the context of hematopoiesis, cytokines—such as thrombopoietin for platelets and erythropoietin for red blood cells—bind to receptors on the surface of hematopoietic stem cells. This binding activates the JAK family of proteins, which in turn activate the STAT proteins. The activated STAT proteins then travel to the cell nucleus, where they bind to DNA and regulate the expression of genes involved in cell survival, proliferation, and differentiation.

Under normal physiological conditions, this pathway is exquisitely regulated. It is only activated when a specific cytokine is present and is quickly turned off to prevent overproduction of blood cells. The driver mutations found in ET (*JAK2*, *CALR*, and *MPL*) fundamentally disrupt this regulation. They cause the JAK-STAT pathway to become constitutively active, sending a relentless and powerful growth signal to the nucleus even in the absence of the normal cytokine stimulation. This abnormal, persistent signaling is the molecular engine of the disease, driving the overproduction of megakaryocytes and the resulting thrombocytosis that defines ET.

Furthermore, this chronic activation of the JAK-STAT pathway contributes to a state of systemic inflammation, which is a hallmark of MPNs. The malignant clone itself, as well as non-malignant cells in the bone marrow microenvironment, produce an excess of inflammatory cytokines (such as IL-8 and TNFα). This inflammatory milieu not only contributes to the debilitating constitutional

symptoms experienced by many patients—such as profound fatigue, night sweats, fever, and bone pain—but is also thought to create a supportive niche that favors the malignant clone over normal hematopoiesis, potentially driving disease progression and fibrotic transformation.

2.4. Predisposition, Clinical Course, and Progression

The clinical presentation and course of ET can be highly variable. The median age at diagnosis is typically around 60 years, though a second peak occurs in younger individuals, particularly women. Many patients—over 50% in some cohorts—are entirely asymptomatic at the time of diagnosis, with the condition being discovered incidentally on a routine complete blood count that reveals a high platelet count. When symptoms are present, they are often related to either microvascular disturbances caused by small platelet aggregates or major thrombotic events. Common symptoms include headaches (including ocular migraines), dizziness, visual disturbances, and erythromelalgia—a characteristic burning pain and redness in the hands and feet.

While most cases of ET are sporadic and arise from random somatic mutations, a clear familial predisposition has been identified. First-degree relatives of individuals with an MPN have a five- to seven-fold increased risk of developing an MPN themselves. This does not imply that the disease is simply inherited in a Mendelian fashion, but rather that an underlying constitutional genetic background can modulate the phenotype and increase susceptibility. This concept is crucial, as it suggests that certain individuals may be more vulnerable to the effects of environmental or occupational mutagens. An external exposure could act as the "second hit" or the critical trigger that initiates the disease process in a genetically predisposed individual.
The primary risks associated with ET are thrombosis and disease progression. The risk of blood clots is stratified based on several well-defined factors: age over 60, a prior history of thrombosis, and the presence of the *JAK2* V617F mutation. Cardiovascular risk factors such as hypertension, diabetes, high cholesterol, and particularly tobacco use, further elevate this risk.
Disease progression represents a significant long-term threat. Over a median of 8.5 years, approximately 10% of patients with ET will progress to myelofibrosis, a condition characterized by severe bone marrow scarring, anemia, and an enlarged spleen. Around 3% of patients will transform to acute myeloid leukemia (AML), a highly aggressive and often fatal blood cancer. Risk factors for these transformations have been identified and include the specific type of driver mutation (*MPL* variants increase fibrosis risk), extreme thrombocytosis, and the acquisition of additional, non-driver mutations over time.

Table 1: WHO Diagnostic Criteria for Essential Thrombocytosis

To provide a standardized medical framework for the condition under review, the 2016 World Health Organization (WHO) diagnostic criteria for Essential Thrombocytosis are presented below. Diagnosis requires meeting all four major criteria, or the first three major criteria and the one minor criterion.

Criteria Type	Diagnostic Criterion
Major	1. Sustained platelet count \geq 450 \times 10^9/L.
Major	2. Bone marrow biopsy showing proliferation

Criteria Type	Diagnostic Criterion
	mainly of the megakaryocyte lineage with increased numbers of enlarged, mature megakaryocytes with hyperlobulated nuclei. No significant increase or left-shift in neutrophil granulopoiesis or erythropoiesis.
Major	3. Not meeting WHO criteria for other myeloid neoplasms such as Chronic Myeloid Leukemia (*BCR-ABL1*+), Polycythemia Vera, Primary Myelofibrosis, or Myelodysplastic Syndromes.
Major	4. Presence of a *JAK2*, *CALR*, or *MPL* mutation.
Minor	In the absence of a *JAK2*, *CALR*, or *MPL* mutation, the presence of another clonal marker, or absence of evidence for reactive thrombocytosis.

Section 3: The Occupational Profile of a Canadian Armed Forces Combat Engineer

3.1. Core Duties and Responsibilities: A Multi-Hazard Profession

The Canadian Armed Forces (CAF) Combat Engineer is a foundational member of the combat arms team, operating under the motto "Ubique," meaning "Everywhere". This motto aptly describes their pervasive presence and critical role in all operational environments. Their primary mission is to enable friendly forces to live, move, and fight effectively on the battlefield while simultaneously impeding the enemy's ability to do the same. This dual mandate requires a uniquely broad and demanding skill set, making the Combat Engineer trade a true "jack-of-all-trades" and inherently a multi-hazard profession.

The core responsibilities of a Combat Engineer can be categorized into three main areas:

- **Mobility and Counter-Mobility:** This involves shaping the physical battlefield. Mobility tasks include the construction of essential infrastructure under austere conditions, such as tactical bridges, roads, airfields, and helicopter landing sites, often using heavy equipment. Counter-mobility tasks are the inverse: creating obstacles to enemy movement through the demolition of bridges and roads and the strategic laying of minefields. A critical and high-risk component of this role, particularly in modern conflicts like Afghanistan, is route clearance—the detection and disposal of land mines, booby traps, and Improvised Explosive Devices (IEDs).

- **Survivability:** Combat Engineers are responsible for enhancing the protection of friendly forces. This includes the construction of field defences, such as bunkers and fortified positions, which often involves extensive earth-moving, carpentry, and work with various

building materials. They are also tasked with providing essential life support, such as sourcing and purifying local water supplies to make them potable.

- **General Engineering and Combat Support:** This broad category encompasses the operation, maintenance, and repair of a vast array of military equipment. Combat Engineers are proficient in the use of weapons, communications equipment, and a wide range of vehicles, from standard military pattern trucks to specialized Armoured Engineering Vehicles (AEVs). They are also trained to fight as infantry when required, underscoring their frontline combat role.

The operational environment for a Combat Engineer is defined by its hazardous nature. Tasks are physically grueling, involving heavy lifting, prolonged work in awkward positions, and endurance under extreme weather conditions. The psychological stress of handling live munitions and operating in combat zones is immense. Furthermore, the work environment is characterized by constant exposure to physical and chemical hazards, including high noise levels, vibration from equipment, and work in confined spaces like vehicle hulls or trenches.

3.2. Training Environments and Associated Exposures

The career of a Combat Engineer begins with intensive and prolonged training that establishes a baseline of chronic exposure to occupational hazards. This training is not a benign classroom activity but a hands-on, physically demanding regimen designed to replicate the conditions of operational deployment. The primary training establishment for this trade is the Canadian Forces School of Military Engineering (CFSME) at CFB Gagetown, New Brunswick, a center of excellence for military engineering.

The training curriculum is broken into modules that directly correspond to their core duties, each with its own set of inherent exposures :

- **Demolitions Training:** This is a fundamental skill for Combat Engineers. Recruits learn to handle, prepare, and detonate a variety of military explosives, such as C4. This training takes place on dedicated demolition ranges where personnel are repeatedly exposed to the constituent chemicals of the explosives through handling and, more significantly, to the toxic gases and particulate residues released upon detonation.

- **Vehicle and Equipment Operation:** Trainees learn to operate and perform basic maintenance on numerous vehicles and pieces of heavy equipment. This phase of training ensures repeated exposure to fuel, oil fumes, and exhaust gases—an exposure explicitly listed in the official MOSID Task Statement for the trade. Dermal contact with fuels, lubricants, and cleaning solvents is a common occurrence.

- **Field Engineering and Construction:** Training exercises involve building structures like Bailey bridges, digging trenches, and constructing field fortifications. These activities generate significant amounts of dust and particulate matter from soil and construction materials. On military training grounds, this dust is not merely inert soil; it can be contaminated with the accumulated residues from decades of live-fire exercises, including heavy metals and unconsumed explosive compounds.

This continuous cycle of training exercises, conducted both at CFSME and at their home units, means that a Combat Engineer's exposure to this specific cocktail of chemical and physical hazards begins on day one of their trade qualification and continues throughout their entire career. It is a foundational and inescapable aspect of the occupation.

3.3. Deployment Profiles: Domestic and Foreign Theatres

The skills honed during training are applied in a variety of operational settings, both within Canada and abroad. Each deployment type carries its own unique exposure profile, often intensifying the hazards encountered during routine training.

- **Domestic Operations (DOMS):** CAF Combat Engineers are a key component of Canada's response to natural disasters and national emergencies, frequently deployed under the banner of Operation LENTUS. They have been instrumental in relief efforts for floods, forest fires, and ice storms across the country. During these operations, their primary tasks include clearing debris, constructing temporary bridges, reinforcing infrastructure, and operating heavy machinery for extended periods. These deployments, while not in a combat zone, involve concentrated and prolonged exposure to hazards such as diesel exhaust from generators and heavy equipment, fuels, oils, and potentially contaminated environmental dust and smoke from widespread fires.

- **Foreign Deployments (Afghanistan):** The CAF mission in Afghanistan from 2001 to 2014 represents a period of intense operational tempo and extreme environmental exposure for Combat Engineers. Deployed to regions like Kandahar, they were at the forefront of combat operations, facing a uniquely hazardous environment. Their roles in this theatre magnified their occupational exposures significantly:

    - **Route Clearance and IED Disposal:** As a primary task, engineers were constantly engaged in clearing roads of IEDs. This involved not only the stress of operating under constant threat but also close and frequent contact with a wide variety of explosive materials, both military-grade and homemade. Disposal, often through controlled detonation, resulted in repeated, close-proximity inhalation of detonation byproducts.

    - **Forward Operating Base (FOB) Life:** Combat Engineers were often the first troops to establish FOBs, living and working on these bases for the duration of their tours. A ubiquitous feature of these FOBs was the use of open-air burn pits for waste disposal. These pits operated 24/7, incinerating a vast array of materials and blanketing the surrounding area in a plume of toxic smoke. Combat Engineers, due to their construction and maintenance roles, were often located in close and sustained proximity to these pits, leading to significant inhalation exposure to a complex and highly toxic mixture of chemicals.

The combination of intensive training, frequent domestic deployments, and arduous combat tours in environments like Afghanistan creates a career-long exposure profile for Combat Engineers that is both chronic and acute, involving a synergistic mix of chemical and physical hazards.

Table 2: Summary of Occupational Duties and Associated Chemical Hazards for CAF Combat Engineers

Core Occupational Duty	Primary Associated Chemical and Particulate Hazards
Demolitions & Breaching (Training & Operations)	Direct handling and inhalation of dust from explosives (TNT, RDX, HMX). Inhalation of detonation byproducts (Nitric Oxides, Carbon Monoxide, unconsumed explosive particulates).
Vehicle & Heavy Equipment Operation/Maintenance	Inhalation of diesel and JP-8 fuel vapors (containing **Benzene**). Inhalation of vehicle exhaust (containing **Polycyclic Aromatic Hydrocarbons (PAHs)**, particulate matter, CO, NOx). Dermal absorption of fuels, oils, lubricants, and solvents.
Route Clearance & EOD (Afghanistan)	Close-proximity exposure to a wide variety of military, commercial, and homemade explosives and their detonation byproducts.
Base Construction & Life (Afghanistan)	Prolonged inhalation of smoke from open-air burn pits (containing **Benzene**, **PAHs**, Dioxins, Furans, Heavy Metals, Particulate Matter).
Domestic Disaster Relief (Op LENTUS)	Concentrated, high-tempo exposure to diesel exhaust from generators, pumps, and heavy equipment (**PAHs**, particulate matter). Exposure to fuels and lubricants.
General Field Engineering & Training	Inhalation of contaminated dust and soil on military training ranges (containing heavy metals, explosive residues).

Section 4: Analysis of Service-Related Toxic Exposures for Combat Engineers

4.1. Part A: Explosives and Their Chemical Constituents

The handling, use, and disposal of explosives are defining activities of the Combat Engineer trade. These materials are not inert substances but are complex, energetic chemical mixtures, many of which possess inherent toxicity. Exposure occurs not only through direct contact but also through the inhalation of dust, fumes, and the byproducts of their detonation.

Chemical Composition

Military-grade explosives are formulated to achieve specific performance characteristics, often by blending different chemical compounds. Those most relevant to a Combat Engineer's duties include :

- **Composition C-4:** A common plastic explosive used for demolition charges. Its primary energetic component is **RDX (hexahydro-1,3,5-trinitro-1,3,5-triazine)**, constituting approximately 91% of its mass. The remainder is a mixture of plasticizers and binders, such as dioctyl sebacate and polyisobutylene, designed to make it malleable and stable.

- **Composition B:** A powerful melt-cast explosive frequently used as the bursting charge in artillery shells, mortars, and grenades. It is a mixture of approximately 60% **RDX** and 39% **TNT (2,4,6-trinitrotoluene)**, with about 1% wax as a desensitizer.

- **TNT (2,4,6-trinitrotoluene):** A foundational military explosive, TNT is a nitroaromatic compound used on its own and as a component in many other explosive mixtures like Composition B and Tritonal.

- **Other Energetic Materials:** The military arsenal includes a wide variety of other compounds that engineers may encounter, including **HMX (octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine)**, a more powerful relative of RDX; **PETN (pentaerythritol tetranitrate)**, used in detonating cords and blasting caps; and various propellants based on **nitrocellulose** and **nitroglycerin**.

Detonation Byproducts and Residues

A chemical explosion is an extremely rapid combustion (oxidation) reaction. Contrary to a common misconception, the process is never 100% efficient. Consequently, the post-detonation environment contains not only gaseous byproducts but also unconsumed particulate residues of the original explosive compounds.

- **Gaseous Byproducts:** The detonation of organic explosives releases a large volume of hot gases, primarily stable molecules like carbon dioxide (CO_2), carbon monoxide (CO), water vapor (H_2O), and nitrogen gas (N_2). However, harmful byproducts are also consistently formed, most notably **nitric oxides (NOx)**, which are known to be produced from the detonation of TNT and other nitrogen-based explosives. These gases are toxic upon inhalation and contribute to the immediate post-blast fume cloud.

- **Particulate Residues:** Studies of live-fire and blow-in-place detonations have consistently shown that a small but significant fraction of the original explosive material is not consumed in the blast but is instead aerosolized and scattered into the environment as fine particulate matter. This means that Combat Engineers operating in a post-detonation environment are exposed to inhalable and ingestible particles of the parent explosive compounds, such as RDX and TNT. The amount of residue is considerably higher in blow-in-place detonations, a common procedure for disposing of unexploded ordnance.

Toxicology and Health Risks

The constituent chemicals of military explosives have well-documented toxicological profiles, with significant implications for hematological health and carcinogenicity.

- **TNT (2,4,6-trinitrotoluene):** The U.S. Environmental Protection Agency (EPA) has

classified TNT as a Group C "possible human carcinogen". Its primary health effects are hematological and hepatic (liver-related). Exposure is strongly linked to **anemia**, including severe **aplastic anemia** (a condition where the bone marrow fails to produce new blood cells), which was a significant cause of death among munitions workers in the World War I era. Chronic exposure in animal studies has been shown to cause bone marrow fibrosis and, in female mice, a statistically significant increase in the incidence of **leukemia and/or malignant lymphoma**. Preliminary epidemiological data from a German population living near former munitions plants also suggest an increased risk of acute and chronic myelogenous leukemia.

- **RDX (hexahydro-1,3,5-trinitro-1,3,5-triazine):** The U.S. EPA has also classified RDX as a Group C "possible human carcinogen" based on evidence of liver tumors in female mice. RDX is a known neurotoxin, with acute exposure causing seizures in both humans and animals. Of paramount relevance to this report, toxicological studies have demonstrated that RDX and its primary metabolite, MNX, are myelosuppressive, meaning they suppress the function of the bone marrow. The evidence indicates that these compounds specifically target an early, multipotential **hematopoietic stem cell**—the very origin cell of MPNs. The resulting toxicity in animal models has been observed to mimic some clinical features of idiopathic myelofibrosis, a related myeloproliferative neoplasm. This provides a direct mechanistic link between RDX exposure and pathology within the hematopoietic stem cell compartment.

Combat Engineers face these risks through multiple pathways: inhalation of dust during the handling and preparation of charges, inhalation of the fume cloud and particulate residues post-detonation, and potential ingestion of contaminated soil or water in training and operational areas.

4.2. Part B: Burn Pit Emissions in the Afghanistan Theatre of Operations

The use of open-air burn pits as the primary method of waste disposal at military bases during the conflict in Afghanistan created a unique and highly hazardous exposure environment for deployed personnel. Combat Engineers, due to their roles in base construction, maintenance, and general operations, were often stationed in prolonged and close proximity to these continuous sources of toxic emissions.

Practice and Composition

Burn pits were used to incinerate a vast and uncontrolled range of waste materials, often using jet fuel (JP-8) as an accelerant. The materials burned included a heterogeneous mix of plastics, rubber, styrofoam, medical and human waste, metal and aluminum cans, petroleum and lubricant products, paints, solvents, and even unexploded ordnance and munitions. This practice, which is banned for municipal waste within the United States due to its known health hazards, generated an estimated 65,000 to 85,000 pounds of solid waste smoke per day at large bases.

Resulting Emissions

The incomplete and low-temperature combustion of this diverse waste stream released a complex aerosol of toxic chemicals and particulate matter into the ambient air of the bases. Air sampling studies conducted at bases in Iraq and Afghanistan have identified numerous hazardous compounds in burn pit smoke :

- **Particulate Matter (PM):** Burn pits were a major source of fine (PM_{2.5}) and coarse (PM_{10}) particulate matter, with average concentrations exceeding U.S. air pollution standards. These fine particles can be inhaled deep into the lungs, cross into the bloodstream, and distribute throughout the body.

- **Volatile Organic Compounds (VOCs):** This class of chemicals includes **benzene**, a known human carcinogen and potent hematotoxin. Air samples from Joint Base Balad, Iraq, showed levels of benzene that exceeded safety thresholds.

- **Polycyclic Aromatic Hydrocarbons (PAHs):** These are a group of potent carcinogens formed during the incomplete combustion of organic materials. PAHs were identified as a significant component of burn pit emissions.

- **Dioxins and Furans:** Polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are highly toxic compounds known to cause cancer. Burn pits were identified as the single most important source of dioxins and furans at Joint Base Balad.

- **Heavy Metals and Other Toxicants:** The smoke contained a variety of other harmful substances, including heavy metals like lead and mercury, oxidized titanium and iron, and toxic intermediates like acrolein.

Health Consequences

Exposure to this toxic mixture has been linked to a wide range of adverse health outcomes. While respiratory and cardiovascular diseases are the most studied, the carcinogenic potential of the emissions is of significant concern. In recognition of the scientific evidence, the United States government, through the PACT Act, has established a presumptive service connection for a long list of diseases for veterans exposed to burn pits in the Southwest Asia theatre of operations. This list includes numerous cancers, and of particular relevance to ET, it explicitly names **acute and chronic leukemias**, **lymphoma of any type**, **multiple myeloma**, and **myelodysplastic syndromes** as presumptive conditions. The inclusion of these other clonal myeloid and hematological malignancies, which share biological pathways and target cells with ET, provides a powerful policy precedent based on the established hazardous nature of burn pit exposure.

4.3. Part C: Pervasive Occupational Exposures

Beyond the acute hazards of explosives and burn pits, Combat Engineers are subject to a range of pervasive occupational exposures throughout their careers, stemming from the constant operation and maintenance of vehicles and equipment. These chronic, often low-level

exposures contribute significantly to their cumulative toxic burden.

Petroleum, Oils, and Lubricants (POLs)

Combat Engineers operate and maintain a fleet of diesel-powered vehicles, from standard trucks to AEVs. This work necessitates constant interaction with military-grade fuels and lubricants.

- **Military Fuels (JP-8 and Diesel):** Military jet fuel (JP-8) and diesel fuel are complex mixtures of hundreds of aliphatic and aromatic hydrocarbons. While their exact composition varies, a key constituent of toxicological concern is **benzene**. Benzene is a natural component of crude oil and is present in these refined fuels. Exposure occurs through the inhalation of fuel vapors during refueling operations and through dermal absorption from spills and contact during maintenance.

- **Benzene Toxicology:** Benzene is classified by the International Agency for Research on Cancer (IARC) as a Group 1 carcinogen, meaning it is definitively **carcinogenic to humans**. Its primary target organ is the bone marrow. Chronic exposure to benzene is a well-established cause of **acute myeloid leukemia (AML)**, **aplastic anemia**, and **myelodysplastic syndromes (MDS)**. This direct, causal link between benzene and other clonal disorders of hematopoietic stem cells is a critical piece of evidence.

Vehicle and Equipment Maintenance

The maintenance of military equipment involves the use of numerous industrial chemicals, further adding to a Combat Engineer's exposure profile.

- **Solvents and Degreasers:** These products are used ubiquitously for cleaning engine parts and equipment. Many industrial solvents are mixtures containing hazardous chemicals, including benzene, toluene, and other VOCs. Exposure is primarily through inhalation of volatile vapors in poorly ventilated maintenance bays and through direct skin contact, which can lead to systemic absorption.

- **Chemical Agent Resistant Coating (CARC) Paint:** Military vehicles are coated with CARC paint, which provides chemical resistance. The painting process, as well as any sanding or grinding of painted surfaces during repair, can release hazardous compounds. The most notable is hexamethylene diisocyanate (HDI), a potent respiratory sensitizer, along with a variety of solvents.

Environmental Particulates

In addition to specific chemicals, Combat Engineers are immersed in environments with high levels of airborne particulate matter from multiple sources.

- **Diesel Exhaust:** The operation of diesel engines in trucks, generators, and heavy equipment is a constant feature of both training and deployment. Diesel exhaust is a complex mixture of gases and fine particulate matter (soot) and is also classified by IARC as a **Group 1 human carcinogen**. A key carcinogenic component of diesel soot is

Polycyclic Aromatic Hydrocarbons (PAHs).

- **Dust and Soil:** Military training and operational areas are inherently dusty environments. This dust is not benign; it can be laden with the residues of munitions (heavy metals, explosive compounds) and other contaminants from military activities, which can then be resuspended and inhaled. While various dust suppressants are used, these are themselves chemical mixtures that can contribute to the overall exposure burden.

The occupational profile of a Combat Engineer is thus defined by a continuous and overlapping exposure to a specific set of hematotoxic and carcinogenic agents. The ubiquity of benzene and PAHs, in particular, from fuels, solvents, and exhaust, creates a chronic exposure risk that persists throughout a member's entire service, from the training base in Canada to the FOB in Afghanistan.

Section 5: The Etiological Link: From Military Exposures to Essential Thrombocytosis

The establishment of a service connection for a disease with a complex etiology like Essential Thrombocytosis requires more than demonstrating exposure to hazardous substances; it necessitates the construction of a scientifically plausible causal pathway that links those exposures to the specific molecular pathogenesis of the disease. The evidence from toxicology, molecular biology, and epidemiology allows for the delineation of such a pathway, connecting the chemical exposures inherent to the Combat Engineer trade directly to the acquired somatic mutations that drive ET.

5.1. Mechanisms of Chemical Carcinogenesis: Inducing Somatic Mutations

The development of MPNs, like most cancers, is understood to be a multi-step process that begins with damage to the DNA of a single stem cell. This initial damage can confer a survival or proliferative advantage, allowing the mutated cell to outcompete its normal counterparts and establish a "clone" of genetically identical daughter cells. This process is known as clonal evolution.

Many of the chemical agents present in the Combat Engineer's environment are classified as genotoxic, meaning they have the capacity to directly damage genetic material. The mechanisms are varied but well-understood:

- **DNA Adduct Formation:** Reactive metabolites of chemicals like benzene and PAHs can covalently bind to DNA, forming structures called DNA adducts. These adducts disrupt the normal structure of the DNA helix. If not repaired by the cell's internal machinery before the cell divides, they can cause errors during DNA replication, leading to permanent changes in the DNA sequence—a mutation.

- **Oxidative Stress:** The metabolism of many toxic chemicals, including benzene, generates reactive oxygen species (ROS) in the bone marrow. ROS are highly reactive

molecules that can attack and damage all components of the cell, including DNA, leading to strand breaks and base modifications that can result in mutations.

- **Chromosomal Aberrations:** Some agents can cause larger-scale damage, such as breaks in chromosomes or the rearrangement of large segments of DNA.

This process of DNA damage and subsequent mutation is the fundamental mechanism by which environmental exposures can initiate cancer. When this damage occurs in a critical gene within a hematopoietic stem cell—such as *JAK2*, *CALR*, or *MPL*—it can provide the precise "on" switch for the JAK-STAT pathway, leading directly to the uncontrolled proliferation that characterizes Essential Thrombocytosis. The disease is, therefore, a direct molecular consequence of the type of cellular damage known to be caused by the chemicals to which Combat Engineers are exposed.

5.2. The Role of Benzene and PAHs in Promoting Clonal Hematopoiesis and JAK2 Mutations

While direct evidence showing that benzene causes the specific *JAK2* V617F mutation is still an area of active research, the circumstantial and mechanistic evidence linking these exposures to the development of *JAK2*-driven neoplasms is exceptionally strong.

Benzene

Benzene is arguably the most well-established chemical hematotoxin and leukemogen. Its primary target is the bone marrow, the site of hematopoiesis.

- **Established Link to Myeloid Neoplasms:** Chronic benzene exposure is a recognized cause of several clonal hematopoietic disorders, including Acute Myeloid Leukemia (AML) and Myelodysplastic Syndromes (MDS). These diseases, like ET, are cancers of the hematopoietic stem and progenitor cells.

- **Direct Link to a *JAK2*-Driven MPN:** Crucially, epidemiological studies have established a direct link between high levels of benzene exposure and the development of Primary Myelofibrosis (PMF). PMF is a classical MPN that shares the same spectrum of driver mutations as ET, with approximately 65% of PMF cases being driven by a *JAK2* mutation. Given that ET, PV, and PMF exist on a biological continuum, are all driven by the same core mutations in the JAK-STAT pathway, and can transform into one another, a known chemical cause for one member of the group strongly implies a potential causal role in the others.

- **Mechanism of Action:** The mechanism by which benzene causes these diseases involves its metabolism in the liver and bone marrow to reactive quinones and aldehydes. These metabolites induce oxidative stress and directly damage the DNA of hematopoietic stem and progenitor cells (HSPCs), the very cell population in which the *JAK2* mutation must arise to cause disease.

Polycyclic Aromatic Hydrocarbons (PAHs)

PAHs, ubiquitous in diesel exhaust and burn pit smoke, represent another class of potent carcinogens that target the hematopoietic system.

- **Induction of Clonal Hematopoiesis:** A pivotal piece of evidence comes from studies of World Trade Center (WTC) first responders, who were exposed to an aerosolized dust cloud containing a high concentration of PAHs and other carcinogens. These studies found a significantly higher incidence of **clonal hematopoiesis (CH)** in the exposed firefighters compared to controls.

- **Clonal Hematopoiesis as a Precursor to MPN:** Clonal hematopoiesis is a recognized pre-malignant state. It is defined by the presence of a detectable clone of blood cells that have all descended from a single stem cell carrying a somatic mutation. Individuals with CH have a substantially increased risk of subsequently developing a hematological malignancy, including MPNs.

- ***JAK2* Mutation in Clonal Hematopoiesis:** Significantly, the *JAK2* V617F mutation is one of the most frequently identified mutations in individuals with asymptomatic clonal hematopoiesis. Studies have shown that the *JAK2* mutation can be acquired early in life, even in utero, and can lie dormant for decades before some subsequent event—such as a potent inflammatory stimulus—promotes the expansion of the clone, leading to a clinical diagnosis of MPN.

This evidence creates a clear and logical pathway: exposure to PAHs, a key component of the Combat Engineer's environment, can induce clonal hematopoiesis. This clonal hematopoiesis is often driven by the *JAK2* mutation. This state is the direct, identifiable precursor to the development of a clinical MPN like Essential Thrombocytosis.

5.3. Epidemiological Evidence: Increased MPN Incidence in Veteran Populations

The mechanistic links established by toxicological and molecular studies are strongly corroborated by large-scale epidemiological studies of military veteran populations. These studies consistently demonstrate that veterans, particularly those who served in the post-9/11 era, face a significantly elevated risk of developing MPNs compared to both earlier veteran cohorts and the general population.

A major retrospective cohort study published in the *American Journal of Hematology* analyzed the health records of nearly half a million U.S. veterans from the Korean War, Vietnam War, and Persian Gulf War eras. The findings were stark:

- **Highest Risk in Persian Gulf War Era Veterans:** Veterans from the Persian Gulf War era (defined as service from 1990 to the present, encompassing deployments to Iraq and Afghanistan) had the highest risk of developing an MPN. Their risk was nearly five times higher than that of Korean War-era veterans and 2.5 times higher than that of

Vietnam-era veterans.

- **Younger Age at Diagnosis:** Persian Gulf War-era veterans were diagnosed with MPNs at a significantly younger age than their counterparts from previous conflicts. This finding is particularly concerning, as MPNs are typically diseases of older age (median age of diagnosis is ~60). An earlier onset suggests that a potent environmental or occupational exposure may have accelerated the disease process.

- **Worse Clinical Outcomes:** Despite being younger, the Persian Gulf War-era veterans with MPNs had higher risks of thrombosis and bleeding and, consequently, lower overall survival rates compared to veterans from earlier wars. This may indicate a more aggressive disease phenotype, potentially driven by the inflammatory environment associated with their service-related exposures.

- **Increased Incidence Rate:** The yearly age-adjusted incidence of MPNs in this veteran cohort was found to be 10.5 per 100,000 population, a rate approximately triple that of the general population.

Another study specifically investigating veterans stationed at Marine Corps Base Camp Lejeune during a period of known water contamination with VOCs, including benzene, found a statistically significant 68% increased rate of myelodysplastic and myeloproliferative syndromes compared to a control group of veterans stationed at a base without contaminated water. These epidemiological findings provide powerful, real-world evidence that the exposures associated with modern military service, particularly those involving burn pits and chemical contaminants like benzene, are associated with a tangible and significant increase in the risk of developing these rare blood cancers.

5.4. Synthesizing the Evidence: A Causal Pathway from Combat Engineer Exposures to ET

The cumulative evidence allows for the construction of a clear, logical, and scientifically sound causal pathway linking the occupational exposures of a CAF Combat Engineer to the development of Essential Thrombocytosis:

1. **Exposure:** A CAF Combat Engineer, through the inherent duties of their trade during training and deployment, experiences chronic and repeated exposure to a synergistic mixture of genotoxic and hematotoxic agents. The most prominent and well-characterized of these are benzene (from fuels and solvents) and PAHs (from diesel exhaust and burn pit smoke).

2. **Initiation (DNA Damage):** These chemicals and their metabolites are absorbed into the body and transported to the bone marrow. There, they directly damage the DNA of long-lived hematopoietic stem cells through mechanisms such as DNA adduct formation and oxidative stress.

3. **Mutation (Clonal Hematopoiesis):** This DNA damage can result in the acquisition of a somatic mutation in a key regulatory gene, most commonly *JAK2*. This mutated stem cell

gains a proliferative advantage and begins to expand, creating a detectable clone of abnormal cells—the state of clonal hematopoiesis.

4. **Promotion (Clonal Expansion):** The chronic inflammatory state induced by both the ongoing exposures and the mutated clone itself creates a bone marrow microenvironment that favors the survival and expansion of the abnormal cells over normal hematopoiesis.

5. **Progression (Clinical Disease):** As the clone expands, it leads to the overproduction of mature blood cells, in this case, platelets. When the platelet count reaches a clinically significant threshold and other diagnostic criteria are met, the individual is diagnosed with Essential Thrombocytosis.

This pathway is not speculative. Each step is supported by evidence from toxicology, molecular biology, and epidemiology. It provides a robust scientific foundation for concluding that the occupational service of a CAF Combat Engineer can be, at the very least, a significant contributing factor to the development of Essential Thrombocytosis.

Section 6: Policy Precedent and Framework for Recognition

The scientific evidence linking military service exposures to the pathogenesis of Essential Thrombocytosis is compelling. To translate this evidence into a basis for disability benefit entitlement, it must be considered within the context of Veterans Affairs Canada's adjudication framework and the policy precedents set by allied nations who have grappled with similar issues for their own veteran populations.

6.1. Review of Allied Nations' Policies: The U.S. Presumptive Model

The United States Department of Veterans Affairs (VA) has developed a robust system of "presumptive service connection" for conditions where a strong association with military service has been established through scientific and epidemiological evidence. Under this model, if a veteran with qualifying service in a specific location and time period is diagnosed with a condition on the presumptive list, the VA concedes that the condition was caused by their service without requiring the veteran to provide direct causal evidence.

This model is highly relevant to the case of ET, particularly in light of recent U.S. legislation. The Sergeant First Class Heath Robinson Honoring our Promise to Address Comprehensive Toxics (PACT) Act of 2022 significantly expanded the list of presumptive conditions related to toxic exposures, most notably from open burn pits in the Southwest Asia theater of operations. Crucially, the U.S. VA now presumes service connection for several clonal hematological malignancies for veterans who served in Afghanistan and other specified locations on or after September 11, 2001. These presumptive conditions include :

- **Acute leukemias**
- **Chronic leukemias**
- **Multiple myeloma**
- **Myelodysplastic syndromes (MDS)**

The inclusion of these specific blood cancers is of profound importance. They are, like ET, clonal disorders of hematopoietic stem cells. The scientific rationale that led the U.S. government to link burn pit and particulate matter exposure to leukemias and MDS is directly applicable to ET. MDS, in particular, shares significant clinical and pathological overlap with MPNs, to the extent that some conditions are classified as MDS/MPN overlap syndromes. The U.S. VA's decision was based on a comprehensive review of scientific literature linking exposure to fine particulate matter (PM_{2.5}), which contains benzene, PAHs, and heavy metals, to these specific cancers. This creates a powerful international policy precedent, demonstrating that a major allied nation, evaluating the same exposures in the same conflict theatre, has already concluded that a causal link to clonal myeloid neoplasms is sufficiently established to warrant presumptive status.

In contrast, the policies of the United Kingdom and Australia are generally less reliant on broad presumptive lists and tend to adjudicate claims on a more individualized, case-by-case basis. However, they maintain robust systems for compensating veterans for any illness or cancer proven to be caused by service. For instance, the UK Ministry of Defence provides specific compensation for mesothelioma, a cancer caused by asbestos exposure during service, acknowledging the link between a specific occupational exposure and a subsequent malignancy.

6.2. Application of Veterans Affairs Canada's Adjudication Principles

Veterans Affairs Canada operates under a legislative and policy framework that is well-suited to adjudicating complex claims like those for ET. The framework provides clear guidance on how to weigh evidence and how to proceed in cases of scientific uncertainty.

Weighing of Scientific Evidence

VAC policy explicitly outlines a structured approach for assessing health-related expert opinions and scientific evidence to determine causality. This process requires an expert to synthesize the available evidence and categorize the strength of the causal link into one of four levels of certainty :

1. **More probable than not that causality exists**
2. **At least as likely as not that causality exists**
3. **Insufficient to form an opinion about causality**
4. **More probable than not that causality does not exist**

For VAC to infer causality, the evidence should meet the threshold of Category 2 or higher. The evidence presented throughout this report—from the molecular mechanisms of chemical carcinogenesis to the specific toxicological profiles of military explosives and the corroborating epidemiological data from veteran populations—is structured to meet and exceed this "at least as likely as not" standard.

The "Benefit of Doubt" Principle

A foundational principle of VAC's adjudication process is the "Benefit of Doubt". This statutory requirement, embedded in both the *Pension Act* and the *Veterans Well-being Act*, compels the

decision-maker to resolve any reasonable doubt in favour of the applicant. The policy states that the adjudicator must draw every reasonable inference in the veteran's favour and must accept any evidence that is credible and uncontradicted.

The case for ET as a service-related condition for Combat Engineers is a quintessential scenario for the application of this principle. The etiology of the disease is complex, the latency period between exposure and diagnosis can be decades, and it is often impossible to precisely quantify the exact dose and duration of chemical exposures that occurred years or decades in the past under arduous operational conditions. In such circumstances, where a strong mechanistic link and supportive epidemiological data exist but absolute certainty is unattainable, the Benefit of Doubt principle is designed to ensure a fair outcome for the veteran. The cumulative weight of the evidence presented in this report is sufficient to create, at a minimum, a reasonable doubt that must be resolved in the applicant's favour.

Table 4: Comparison of Presumptive Conditions for Hematological Malignancies Among Allied Veteran Affairs Departments

Hematological Malignancy	Canada (VAC)	United States (VA)	United Kingdom (MOD)	Australia (DVA)
Leukemias (Acute & Chronic)	Case-by-case	**Presumptive** for Burn Pit/PM Exposure ; Agent Orange	Case-by-case	Case-by-case
Multiple Myeloma	Case-by-case	**Presumptive** for Burn Pit/PM Exposure ; Agent Orange	Case-by-case	Case-by-case
Myelodysplastic Syndromes (MDS)	Case-by-case	**Presumptive** for Burn Pit/PM Exposure ; Agent Orange	Case-by-case	Case-by-case
Myeloproliferative Neoplasms (ET, PV, PMF)	Case-by-case	Case-by-case (Myelofibrosis is presumptive for Burn Pit/PM exposure)	Case-by-case	Case-by-case

Section 7: Conclusion and Recommendations

7.1. Summary of the Compelling Case for Service-Related Connection

The evidence presented in this report constructs a robust, multi-layered argument for the recognition of Essential Thrombocytosis as a condition arising from military service for Canadian Armed Forces Combat Engineers. The case rests on a clear and logical progression of established scientific facts.

First, the nature of the disease itself opens the door to an environmental and occupational etiology. Essential Thrombocytosis is a blood cancer fundamentally caused by *acquired* somatic mutations in hematopoietic stem cells. This is not a condition primarily dictated by inheritance, but one initiated by DNA-damaging events that occur during a person's lifetime.

Second, the nature of the occupation places CAF Combat Engineers in a unique position of risk. Their duties, throughout both training and deployment, ensure chronic and repeated exposure to a synergistic cocktail of chemicals with known genotoxic and hematotoxic properties. Pervasive exposure to benzene from fuels and solvents, polycyclic aromatic hydrocarbons from diesel exhaust, the chemical constituents (RDX, TNT) and detonation byproducts of military explosives, and the complex toxic emissions from open-air burn pits in Afghanistan constitutes a career-long, multi-faceted assault on the bone marrow.

Third, a clear mechanistic pathway connects these specific exposures to the molecular origins of ET. Benzene is a confirmed human carcinogen that targets hematopoietic stem cells and is linked to other *JAK2*-driven myeloproliferative neoplasms. PAHs are known to induce clonal hematopoiesis, the direct pre-malignant state from which MPNs arise. Toxicological data on RDX show that it, too, targets hematopoietic stem cells and can produce effects that mimic myeloproliferative disorders.

Finally, this mechanistic link is validated by real-world data. Large-scale epidemiological studies of U.S. veterans who served in the same modern conflicts have demonstrated a significantly higher incidence of MPNs, an earlier age of onset, and worse clinical outcomes. This trend strongly suggests an environmental or occupational cause. The policy decisions of the U.S. Department of Veterans Affairs to grant presumptive status to closely related myeloid neoplasms based on these same exposures provide a powerful international precedent, indicating that the scientific evidence is sufficient to warrant state recognition.

When considered in its totality, the evidence meets and exceeds the "at least as likely as not" standard of proof required by Veterans Affairs Canada. The inherent complexities of the disease's long latency and the difficulty in quantifying historical exposures make this a clear case for the application of VAC's foundational "Benefit of Doubt" principle.

7.2. Formal Recommendation to Veterans Affairs Canada

In light of the comprehensive scientific, toxicological, epidemiological, and policy evidence detailed in this report, it is formally recommended that Veterans Affairs Canada take the following actions:

1. **Recognize Essential Thrombocytosis as a Service-Related Condition:** It is recommended that VAC formally recognize Essential Thrombocytosis—and by logical extension, the other classical Philadelphia-negative Myeloproliferative Neoplasms (Polycythemia Vera and Primary Myelofibrosis) that share an identical molecular pathogenesis—as a condition for which a causal link to military service can be established.

2. **Establish an Adjudication Framework for High-Risk Trades:** This recognition should, at a minimum, create a streamlined adjudication framework for claims submitted by

veterans from high-risk trades, such as Combat Engineer. This framework should give significant weight to the body of scientific evidence linking the characteristic exposures of the trade to the development of MPNs.

3. **Apply the Benefit of Doubt Principle:** Decision-makers should be directed to apply the Benefit of Doubt principle to its fullest extent in these cases, acknowledging the long latency periods and the inherent difficulties in providing precise exposure dosimetry from past service. A veteran's credible statement of exposure, combined with a confirmed diagnosis and a plausible service history, should be considered sufficient to meet the evidentiary threshold.

4. **Consider Presumptive Service Connection:** It is further recommended that VAC give serious consideration to establishing a presumptive service connection for Essential Thrombocytosis and other classical MPNs for veterans with qualifying service in the Southwest Asia theatre of operations (including Afghanistan), in alignment with the policies of allied nations for similar hematological malignancies and exposures.

Adopting these recommendations would ensure that Canadian veterans who develop these serious, chronic blood cancers as a result of their dedicated and hazardous service receive the timely benefits, support, and recognition that they have earned and deserve.

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