Pain and Pleasure in the Motivation-Emotion-Cognition Loop: Robots as Tools and Models

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"Je suis de ceux qui pensent que la science a une grande beauté. Un savant dans son laboratoire n'est pas seulement un technicien ; c'est aussi un enfant placé en face de phénomènes naturels qui l'impressionnent comme un conte de fées."

Irène Joliot-Curie
prix Nobel de Chimie (1935), Sous-secrétaire d'Etat à la Recherche (1936)

A ma mère et à mon père, merci, des mots ne sauraient jamais exprimer la gratitude que je ressens face à votre soutien indefectible sans lequel je ne serai pas là aujourd'hui. Je dédie ce travail à mes parents, à ma famille et à mes amis, merci pour votre soutien constant depuis toutes ces années.

Je dédie ce travail à la science, à celles et à ceux qui la font vivre et qui tentent de rendre le monde meilleur en enlevant au malheur de ce monde.

A Lino, puisses-tu grandir dans ce monde meilleur.

•••

To my mother and father, thank you. Words can never express the gratitude I feel for your unwavering support, without which I would not be here today.

I dedicate this work to my parents, to my family and my friends, thank you for your constant support over the years.
I dedicate this work to science and to those who keep it alive and strive to make the world a better place by alleviating the suffering in it.

To Lino, I hope you'll grow in this better world.

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Science is a collective adventure, so this thesis was.

Thank you all.

Abstract

In this thesis, I explore the integration of pain, its perception, its features, and its sensory process into robotic models, focusing on its influence on motivation-based action selection architecture. Drawing inspiration from clinician psychology, neurobiology, and computation neuroscience, I aim to provide a framework with different perspectives to study how bio-inspired pain mechanisms can affect decision-making systems.

Pain plays a crucial role in biological systems, influencing behaviors essential to survival and maintaining homeostasis, yet it is often neglected in emotional models. In humans and other animals, pain serves as an adaptive response to noxious stimuli, triggering protective actions that prevent harm and promote recovery. This thesis seeks to improve action selection by incorporating pain and its related features into robots, extending the current understanding of artificial agents and exploring how robots can use pain to modulate behavior, adapt to threats, and optimize survival.

Embracing the embodied Artificial Intelligence paradigm and building upon prior work on motivation-based action selection models, this thesis proposes to study different perspectives on pain and its impact on action selection.

First, I provide an overview of related work and the state of the art in relevant disciplines.

In the initial part of this work, I propose an enhanced motivation-based action selection architecture by introducing an embodied model that enables robots to perceive and respond to noxious stimuli. Using artificial nociceptors, I simulate the sensation of damage in robotic agents and compute the emotional state of pain as an artificial hormone. This model investigates how varying levels of pain perception influence behavioral responses, with results emphasizing the adaptive value of pain modulation in action selection, particularly in extreme or hazardous environments.

Next, I introduce an artificial hormonal neuromodulation mechanism featuring a simulated cortisol hormone that modulates the action selection process. This cortisol mechanism incorporates temporal dynamics, resulting in habituation and sensitization processes. I demonstrate how hormonal neuromodulation can lead to emergent behaviors that improve the overall response of robotic agents to environmental variability in extreme scenarios.

Additionally, I propose a novel framework for tactile sensing in mobile robotic platforms. This framework computes a nociceptive and mechanoceptive process capable of localizing and classifying noxious and tactile stimuli. In collaboration with Raphaël Bergoin, we send this sensory signal to a spiking neural network, demonstrating the segregation of cortical areas for nociceptive and mechanoceptive signals and learning embodied sensory representations.

Finally, I present an integrated action selection architecture that combines these new mechanoceptive and nociceptive sensory processes, behavioral responses, hormonal neuromodulation, and the learning of embodied representations. This architecture is examined in a social context with varying levels of interaction with predators. I highlight the importance of social interaction in learning embodied sensory representations and demonstrate how this cortex-based model improves hormonal management and action selection in dynamic environments.

In conclusion, I discuss the results of this research and offer perspectives for future work.

Keywords : Emotion modeling, Robotics and neuroscience, Embodied Artificial Intelligence, affective computing, bio-inspired robotics, neurocybernetics

Résumé

Dans cette thèse, j'explore l'intégration de la douleur, sa perception, ses caractéristiques et son processus sensoriel dans des modèles robotiques, particulièrement dans des architectures motivationelles de sélection de l'action. En m'inspirant de la psychologie clinique, de la neurobiologie et des neurosciences computationelles, je souhaite fournir un cadre avec différentes perspectives pour étudier comment des mécanismes bio-inspirés de douleur peuvent affecter la sélection de l'action.

La douleur joue un rôle crucial dans les systèmes biologiques, influençant les comportements essentiels à la survie et au maintien de l'homéostasie, mais elle est souvent négligée dans les modèles émotionnels. Chez l'Homme et les autres animaux, la douleur sert de réponse adaptative aux stimuli nocifs, déclenchant des actions qui protègent contre les dommages et favorisent la guérison. L'objectif de cette thèse est d'améliorer la sélection de l'action en incorporant la douleur et ses caractéristiques associées dans des robots, en élargissant la compréhension actuelle des agents artificiels et en explorant comment les robots peuvent utiliser la douleur pour moduler le comportement, s'adapter aux menaces et optimiser la survie.

En adoptant le paradigme de l'intelligence artificielle incarnée et en s'appuyant sur des travaux antérieurs sur des modèles de sélection de l'action basés sur la motivation, cette thèse propose d'étudier différentes perspectives autour de la douleur et de son impact sur la sélection de l'action.

Dans la première partie de ce travail, je propose une architecture améliorée de sélection de l'action basée sur la motivation en introduisant un modèle incarné qui permet aux robots de percevoir et de répondre à des stimuli nocifs. En utilisant des nocicepteurs artificiels, je simule la sensation de dommage chez les agents robotiques et calcule leur état émotionnel de douleur en tant qu'hormone artificielle. Ce modèle étudie comment différents niveaux de perception de la douleur influencent les réponses comportementales, avec des résultats soulignant la valeur adaptative de la douleur dans la modulation de la sélection de l'action, en particulier dans des environnements extrêmes ou dangereux.

Ensuite, je présente un mécanisme de neuromodulation hormonale artificielle, mettant en œuvre une hormone cortisol simulée qui module le processus de sélection d'action. Ce mécanisme de cortisol simulée intègre une dynamique temporelle, ce qui entraîne des processus d'accoutumance et de sensibilisation. Je démontre comment la neuromodulation hormonale peut conduire à des comportements émergents qui améliorent la réponse globale des agents robotiques à la variabilité environnementale dans des scénarios extrêmes.

De plus, je propose un nouveau cadre pour la détection tactile dans les plateformes robotiques mobiles. Ce modèle calcule un processus nociceptif et méchanoceptif capable de

localiser et de classer les stimuli tactiles et nocifs. En collaboration avec Raphaël Bergoin, nous envoyons ce signal sensoriel à un réseau neuronal à spikes, démontrant la ségrégation des zones corticales pour les signaux nociceptifs et méchanoceptifs et l'apprentissage de représentations sensorielles incarnées.

Enfin, je présente une architecture intégrée de sélection de l'action qui combine ces nouveaux processus sensoriels mécanoceptifs et nociceptifs, les réponses comportementales, la neuromodulation hormonale et l'apprentissage de représentations incarnées. Cette architecture est examinée dans un contexte social avec différents niveaux d'interaction avec des prédateurs. Je souligne l'importance de l'interaction sociale et des expériences au début de la vie dans l'apprentissage des représentations sensorielles incarnées et je démontre comment ce modèle basé sur le cortex améliore la gestion hormonale et la sélection de l'action dans des environnements dynamiques.

Mots-clés : modélisation des émotions, robotique et neurosciences, Intelligence Artificielle incarnée, Informatique affective, bio-inspired robotics, neurocybernetics

List of publications

In preparation:

- The importance of early-life pain perception in the construction of embodied representations
- Exploring cortisol-modulated pain perception with simulated hormones

Published:

- The effects of stress and predation on pain perception in robots, L L'Haridon, L Cañamero, proceedings of 2023 11th International Conference on Affective Computing and Intelligent Interaction (ACII), 2023 (IEEE)
- The Emergence of a Complex Representation of Touch Through Interaction with a Robot, L L'Haridon, R Bergoin, BS Bal, M Abdelwahed, L Cañamero, proceedings of International Conference on Simulation of Adaptive Behavior, 106-117, 2024 (Springer)
- Wellbeing and the adaptive value of pain, L l'Haridon, AC de C Williams, L Cañamero, AR4W: Affective Robotics for Well-being , 2022, Workshop of 2022 10th International Conference on Affective Computing and Intelligent Interaction (ACII), 2022 (IEEE)

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Chapter 1

Introduction

1.1 Introduction

Action selection, or making a decision regarding which behavior to execute next in a given situation, is a crucial process for both biological agents and autonomous robots in order to survive and interact with others [5, 6]. Affective processes, such as emotions, play an important role in this decision-making process [7]. Pain is one of this affective phenomena which is too often neglected in artificial and robot models. Embracing the embodied Artificial Intelligence paradigm [8, 9, 10] in which interaction with other agents and the environment [11], I investigate and propose a model of how pain perception can influence the action-selection process from different perspectives. Drawing inspiration from ethology-inspired motivation-based architecture [12], I propose to study pain perception and its related mechanisms from a macroscopic behavioral impact to a more microscopic perspective, studying neurobiology mechanisms with artificial hormones and delving into neuronal mechanisms related to pain. I aim to offer a new model to emotionally modulated action-selection architectures with these perspectives.

In biological systems, pain is essential to regulating behaviors and maintaining internal homeostasis. This affective experience is crucial in guiding decision-making processes, allowing organisms to avoid harm and seek positive stimuli [13] . As part of the motivation-emotion-cognition loop, emotion is part of the bio-regulatory system that modifies perception and action selection, influencing how individuals evolve and interact with their environments. Pain is crucial in survival-related environments as it alerts us to potential threats and physical damage and allows us to select the appropriate behavior to avoid harm, survive, and recover.

The development of a robot model capable of perceiving and responding to pain perception presents opportunities not only for improving autonomous decision-making in robots but also for contributing to the understanding of biological and psychological pain mechanisms. This thesis aims to explore the integration of pain into robotic architecture.

Bio-inspired models that takes inspiration from natural mechanisms allow us to conceive and artificial systems more efficiently: such models are often more straightforward and more efficient in their computational cost [14]. In contrast to heavy computational cost solutions such as deep learning, bio-inspired solutions exploit bio-inspired architectures

and mechanisms, such as local learning mechanisms such as Hebbian learning, which necessitate fewer computational resources. These systems aim to adapt autonomously to dynamic environments with less complex algorithms which are embodied in light robotic platforms, where computational cost and energy consumption matter. This approach allows to develop more resilient robotic platforms with lower ecological footprint.

Pain perception is inherently complex, involving a combination of sensory, emotional, and cognitive components. Pain is typically associated with physical damage, influenced by past experiences, internal states, and environmental cues. This multifaceted nature of pain presents unique challenges for robot modeling. By developing computational models of the neural and hormonal mechanisms underlying pain perception, we can begin to explore how robots might use these signals to adapt their behavior, much like biological organisms.

This research contributes to the interdisciplinary field of affective robotics, bridging the gap between cognitive robotics, robotics, and neuroscience. The objective of this thesis is twofold: first, to use robots as tools to simulate and test theories of pain derived from psychology and neuroscience, and second, to develop better action-selection algorithms using pain perception mechanisms. To achieve this, I focus on the following **research questions**:

- How can pain be modeled in robotic systems to influence action-selection?
- What are the neurobiological and behavioral foundations of pain perception that can apply to robotic architectures?
- How do these models contribute to embodied representations in robots?

1.2 Overview

In this thesis, I explore how pain can model action selection using different perspectives. Building on a motivation-based action-selection architecture, I explore the behavioral impact of pain perception. I propose hormonal neuromodulation of pain perception, I propose a new sensory process and the neural cortex to construct embodied representations of painful stimuli, and finally, I propose a global architecture that includes all these elements. I summarize in Fig. 1.1 the final architecture I propose at the end of this manuscript.

First, in **Chapter 2**, I give an overview of related work and state of state of the art in the different disciplines to which this work contributes.

In **Chapter 3**, I describe a basic action-selection architecture and study how pain perception from a behavioral point of view can affect robot viability in a survival-related problem. I propose a first model of pain perception using simulated damage. I address the question of how our pain system can be adaptive or maladaptive in "modern" or "ancestral" environments with different levels of predator-related danger. This work has been presented in an abstract and poster at the 2022 Affective Robotics For Well-being (AR4W) workshop [15] for the 2022 Affective Computing & Intelligent Interaction (ACII 22).

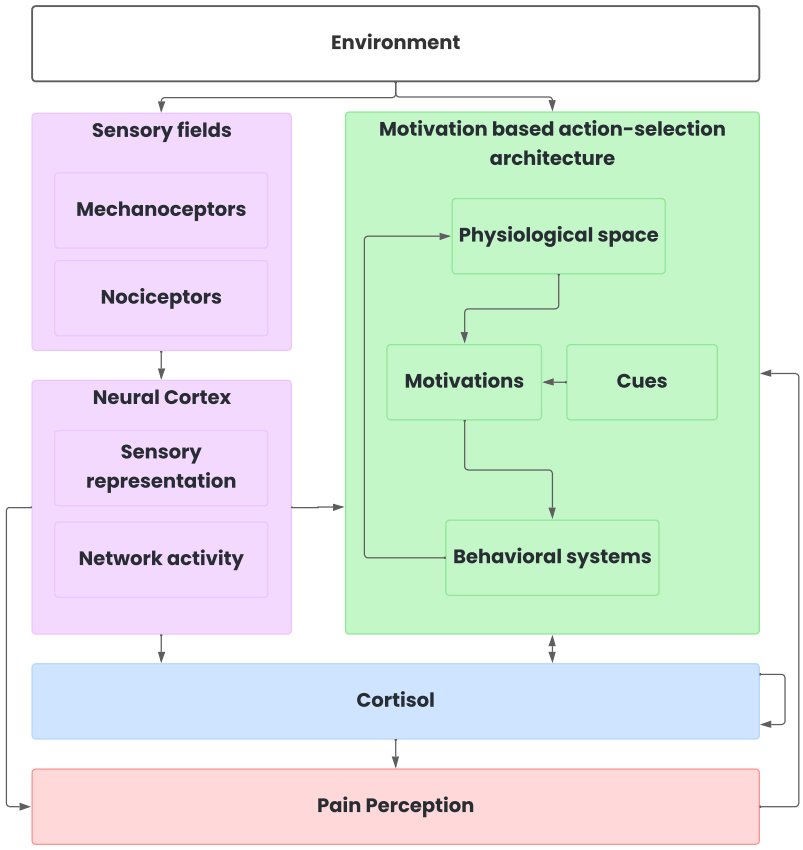


Figure 1.1: High-level diagram of our model and the effects I investigate in different chapters. This overview represents the final model presented in Chapter 6. We will study Green elements in Chapter 3, Blue elements in Chapter 4, magenta elements in Chapter 5, and red elements across all chapters.

Then, in **Chapter 4**, I investigate how neurobiological inspiration can modify this action-selection architecture. Using artificial cortisol hormone, I study how this more complex pain model of perception will influence action selection. I also introduce temporal elements with cortisol neuromodulation, leading to habituation and sensitization mechanisms. This work has been presented and published at the 2023 Affective Computing & Intelligent Interaction (ACII 23) conference [16].

In **Chapter 5**, I investigate the development of body representation. In order to do this, I propose a new model of artificial nociception and mechanoception built on "sensory fields". Using a spiking neural network, I send these new sensory inputs to a simulated cortex. I observed how this simulated cortex would segregate to represent different brain regions specialized in different features of sensory perception. This computational neuroscience approach will allow us to propose a model of unsupervised construction of embodied sensory representation. This work has been presented and published at the 17th International Conference on the Simulation of Adaptive Behavior (SAB 2024) [17].

Building on these works, in **Chapter 6**, I propose an action-selection model with behavioral impact, artificial hormone modulation, and the unsupervised construction of body representation. This gives us a global overview of how pain perception can influence

action selection in a survival problem. I observe how different environments can influence hormonal levels and learned representations and how our model's characteristics can improve survival-related features.

Finally, I conclude this thesis by discussing all of our results and highlighting some of them to discuss how pain perception, from a behavioral, neurobiological, or computational neuroscience perspective, can influence action selection.

1.3 Contributions of the research

The research I propose in this thesis makes the following original contributions:

- A more complex conceptualization of pain in a robot model as not simply a consequence of damage but an emotional state with its sensory process, features, and impact on action selection.
- Experimental investigation of pain (with robots) as an emotional state that modulates behavioral response to noxious stimuli and interaction with other agents and a neuromodulator of embodied action selection architecture (Chapters 3, 4, 6).
- Implemented model of interaction between simulated cortisol and pain perception in an decision making (Chapter 4).
- A novel and original model of artificial nociceptors as a way to process noxious stimuli based on sensory perception of mobile robotic platforms (Chapter 3,4).
- A new framework for tactile representation, with "sensory fields" that allows us to reconstruct complex information about tactile interaction with robot and to propose a model of artificial nociceptors and mechanoceptors for mobile robots (Chapter 5).
- The use of artificial spiking of a neural network as a way to construct complex embodied sensory representation for the mobile robotic platform (Chapters 5, 6).

Chapter 2

State of the Art

2.1 Emotions

Pain is an important component of emotions, and thus it is essential to understand emotions and their representations. The study of emotions has roots in pre-modern history, with texts from Aristotle and Plato and philosophical ideas from Buddhism and Hinduism. However, in the 20th century, building on the 19th-century work of Darwin and James, the study of emotions became a scientific field. As psychology split from philosophy, the study of emotions entered the scientific domain, getting a structured framework and a rigorous methodology. This investigation has continued, focusing on the cognitive, neurobiological, and neuroscientific mechanisms and components of emotions.

Greek philosophy theories are the roots of modern psychology. For Platon, emotions are a complex interplay of sensations, desires, and judgments that face reason. The Platonician dialogs of "Le Timée" and "The Republic" are quoted to illustrate how Platon perceives internal fights between reason and emotions. For Aristotle, Emotions have an important role in action selection and ethics; they are psychological states that, with pain and pleasure, influence moral judgments. We can note that these early studies and emotions face the limit of sociologic context; for example, anger can be defined as a desire for revenge, which may not be sufficient for a universal definition but is rather a characteristic in honor-based societies, whereas vengeance plays a crucial role [18].

Around 200 A.D., Bharata Muni, in the "Natyashastra," a treatise on performing arts, described a dimensional model of emotions known as the 9 "Rasas," [19] which forms a significant foundation for the Indian system of emotion characterization. These Rasas are a superposition of dominant, transitory, and temperamental states. The 9 Rasas in Indian performing arts are Sringaram (erotic), Karunayam (pathetic), Hasyam (comic), Veeram (heroic), Raudram (furious), Bibhatsam (odious), Bhayanakam (terrible), Ad Bhutan (marvelous), Santam (peace).

2.1.1 Dimensional models of emotions

Rather than some preliminary definitions, it is some centuries later that emotional concepts have roots in Spinoza's Ethics [20]. For Spinoza, three elements are sufficient to

describe all emotional states: joy, sadness, and desire. This first definition offers a first view of a dimensional model that defines emotional space.

Wundt [21] further developed this concept with his tri-dimensional theory of emotions (Fig. 2.1), where emotions are viewed as patterns or combinations of emotional state activations, each of which can be characterized by a position within this tri-dimensional space.

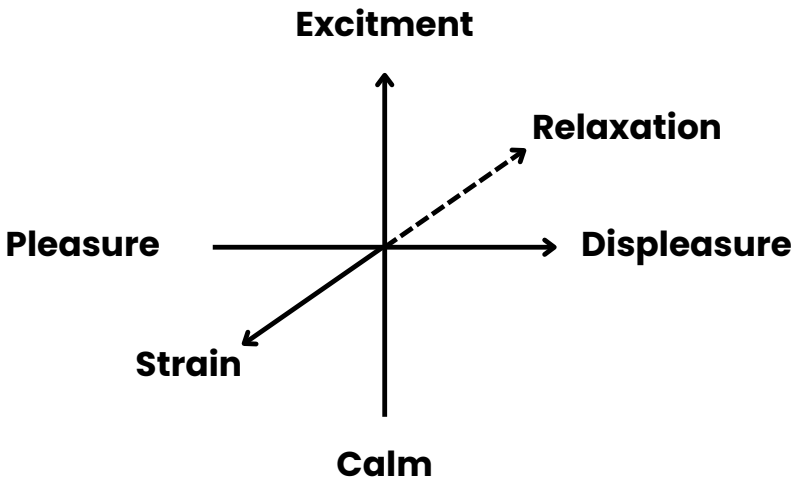


Figure 2.1: Wundt's tridimensional theory of emotion.

Wundt's definition includes the idea of a pleasure/displeasure axis and another of calm /excitement. The displeasure/pleasure axis is often referred to as "valence." Later, a theory of activation (or arousal) tends to define this idea of physiological "activation."

The arousal theory emerged from the works of various people, such as Robert Yerkes and John Dodson, Elizabeth Duffy, and Donald Hebb. It suggests that varying levels of physiological activation drive emotional and cognitive responses.

Yerkes and Dodson introduced the Yerkes-Dodson Law early [22], which proposes the idea that there is an optimal level of arousal for task performance. Moderate arousal leads to peak performance, while too little or too much alters it. Hebb extended this idea by adding cortical activation to this definition, proposing that moderate arousal improves and optimizes learning and cognitive functioning. Duffy proposed arousal as a generalized continuum [23, 24]. This continuum has an emotional intensity varying by activation level. Lindsey linked neurophysiological processes to arousal, specifically in the role of the reticular activating system, emphasizing that arousal is the basis of emotional and behavioral responses across various mental states.

After the first tri-dimensional definition (pleasure-displeasure, dominance-submissiveness, degree of arousal) in 1977 [25], James Russel proposed a "Circumplex" [1] model of effect (Fig. 2.2). Russell proposed a two-dimensional space defined by a degree of arousal and valence axes. He used this space to ask several subjects to classify affect words and emotional states. An inter-individual uniformity in subjects' responses is argued to validate this thesis. Russel argued that we can classify emotions by the angle in the circle

defined by the two dimensions rather than with the actual coordinates in the defined space. This can lead to a one-dimensional definition of emotions.

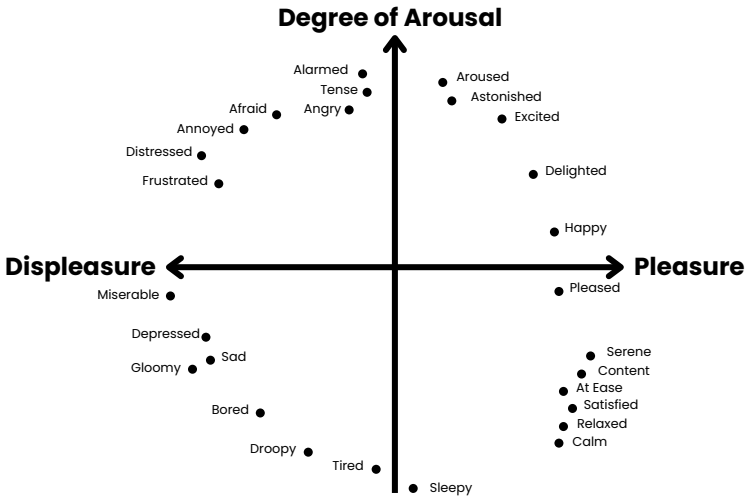


Figure 2.2: Russel's Circumplex model adapted from [1]. 28 affect words are placed in a two-dimensional space: pleasure-displeasure, valence (horizontal), and degree of arousal (vertical). In this space, a circle can characterize pretty uniformly the affect words.

Although many other dimensional definitions of emotional experience exists, there is a general consensus about the valence and arousal dimensions which are the most frequently used.

What characterizes the dimensional approach globally is that every emotional state can be distinguished and qualified on quantitative elements and reactions within the emotional experience. Individual and inter-individual variability of emotional experiences is crucial to dimensional models.

This dimensional approach opposes a discrete basic emotion, which stands by the idea that emotions are distinguished by different reactions that are qualitatively different by nature.

2.1.2 Discrete Basic emotions

In "Les Passions de l'Ame" [26], Descartes distinguished six fundamentally distinct passions: wonder, love, hatred, desire, joy, and sadness. Every emotional experience would be defined as either one or a combination of these six primitive passions. If these 6 passions are no longer commonly shared and use an exhaustive list of primitive emotions, the theory of basic discrete emotions builds on this theory and general idea.

Later, Duschenes stated that there are over 60 different discrete emotions by experientially electrically stimulating a subject's face and reproducing dissecting facial expressions [27]. These experiments relied on the idea that subjects physically expressed emotions through facial expressions.

Darwin further argued in "The Expression of the Emotions in Man and Animals" that emotions are innate and biologically-based responses that serve adaptive functions and are universally shared across humans and even some animals.

James-Lange's theory of emotion [28] states that we do not feel an emotion and then experience a bodily response (like increased heart rate or sweating). But it's our bodily reactions that happen first, and the emotion is then experienced as a consequence of our reaction to these physical changes.

Ekman proposed his Basic Emotions Theory, in which he identified six core emotions: happiness, sadness, fear, anger, surprise, and disgust. These emotions are universal across cultures and identified through distinct facial expressions [29], underlying Darwin's innate character of emotions. He proposed the Facial Action Coding System (FACS) [30] to analyze and categorize humans' facial expressions of emotions.

Tomnkis proposed the Affect Theory [31, 32]. He states that emotions (or affects) are the main drive of human behaviors, more than physiological needs such as thirst or hunger. He proposed nine core affects: excitement, fear-terror, anger-rage. He believed these core affects were innate and linked to facial expression and physiological responses.

Plutchnik offered a model to classify emotion [33], which is a hybrid of both dimensional theory and basic discrete theories. He offers a three-dimensional model (Fig. 2.3) where emotions are arranged in concentrated circles. Primary emotions are in the inner circle, while the more complex ones are in the outer circles. Outer circle emotions are formed by blending inner circle emotions. Plutchnik proposes eight basic emotions that can be expressed in several levels of intensity, represented in the wheel by less intense colors. Emotions opposed in the circle are opposite emotions (e.g., joy and sadness). Complex emotions can be formed using two different emotions; they are called "diads" with several levels of separation of emotions: "primary," one petal apart (e.g., joy and trust give love), "secondary," two petals apart (e.g., anger and sadness give envy) and "tertiary," three petals apart (e.g., fear and disgust give shame). Plutchik's model emphasizes the interconnectivity of emotions, highlighting their crucial role in survival and social interactions.

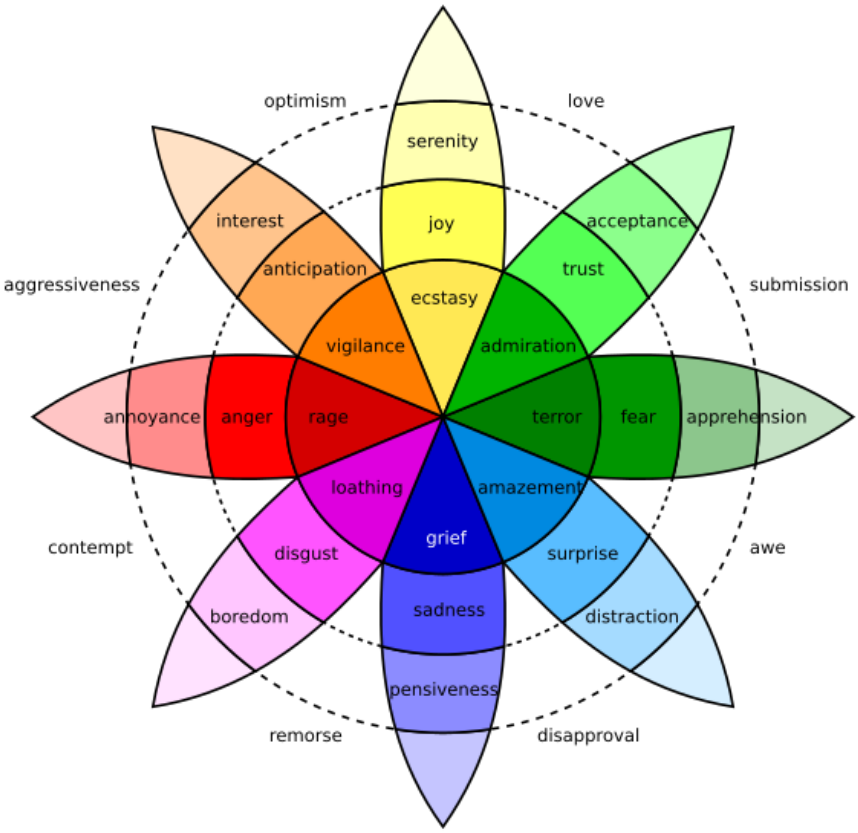


Figure 2.3: Plutchik emotional wheel.

2.1.3 Modern theories

These classic theories have been challenged by modern theories that take a more cognitive perspective. These affective neuroscience theories highlight the crucial role motion plays in biological systems. Motion is part of the bioregulatory system that contributes to the survival, adaptation, and social interaction of both humans and animals.

Jaak Panksepp, in "Affective Neuroscience," [34] identified the brain's basic emotional systems shared by animals and humans. These systems build on neural circuits in the area of the brain and are the basis of emotional experience. This work underlies the biological and, more specifically, neuroscience roots of emotions and leads to many further studies in these circuits, such as seeking, fear, rage, and play systems.

Joseph LeDoux highlighted some neurological mechanisms in "The Emotional Brain" [35] and, in particular, the role of the Amygdala in the process of emotional responses. He argued that emotions, specifically survival-oriented, are deeply embedded in brain circuitry. He described two pathways for processing emotional stimuli: high and low road (Fig. 2.4). The low road is a fast and automatic pathway that sends information directly from the thalamus to the Amygdala for quick reactions. The high road is slower, allowing the cortex to process the stimulus thoroughly before sending it to the Amygdala, allowing a more refined response. This dual system proved a balance between rapid survival

instinct and emotional evaluations.

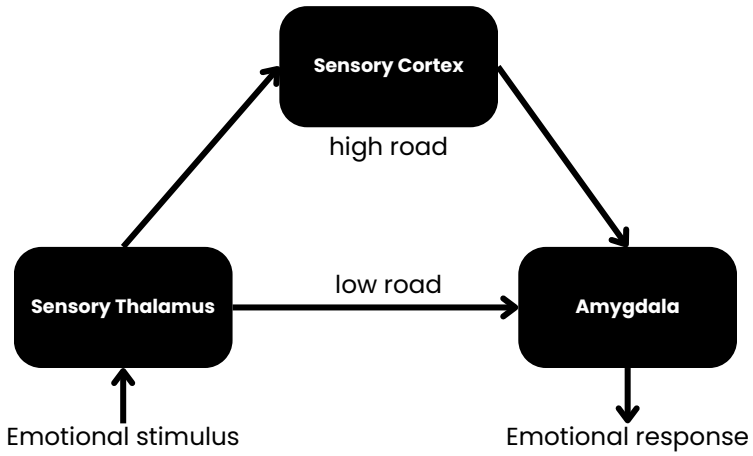


Figure 2.4: The Low and the High Roads to the Amygdala.

Antonio Damasio argued in "Descartes' Error" [7] that emotions are essential for decision-making. He stated that somatic markers (i.e., bodily responses) and emotions guide our choices by creating emotional signals that influence and inform the cognitive process. He highlighted the necessary interaction between emotions, body, and cognition in the context of action selection.

In "How Emotions Are Made," Lisa Feldman Barrett [36] proposes a constructivist theory of emotion that challenges the idea of universal emotions. She argues that emotions are constructed by the brain based on past experience, culture, and context. So, emotions would not be hardwired in the brain; instead, learned concepts are built by predictions and cognitive processing. This is a shift from classical theories, where emotions can be classified into fixed categories.

All these theories of emotions empathize with the crucial role of behavioral response, neurobiological processes, and brain circuitry in emotional responses to stimuli. These are the theories in which "affective computing" or emotional robotics builds.

2.2 Pain

Pain is not the opposite of pleasure neither the negative extreme of valence, but rather an important and often neglected affective component in emotional models. Pain is a crucial element for survival-related action-selection and social interaction. It is an unpleasant sensory and emotional experience linked to real or potential tissue damage; its experience is altered by past experience and relies on complex sensory information [13]. As we propose robotic work, robots or artificial agents cannot "experience" emotional states, but by building on definitions and biologically inspired mechanisms, we can propose a

model of pain perception. This model shall also build on an understanding of the sensory pathway that induces pain.

Pain definition evolved around time since Galen (200 AD) proposed to view it as a disturbance or injury to the body's balance of the four humors: blood, phlegm, yellow bile, and black bile. Pain was seen as an emergence of physical anomalies such as disease or injuries. Later, Descartes Gave the first neurophysiological definition of pain, moving away from the humoral explanation and introducing the central nervous system's role in pain experience. Pains have since been discussed, defined, and studied to give a better understanding of why and how we feel pain.

IASP introduced a revised definition of pain in 1979: "an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage" [37]. They also expanded this definition with six keynotes for additional valuable context.

- Pain is always a personal experience influenced by biological, psychological, and social factors.
- Pain and nociception are different phenomena. Pain cannot be inferred solely from activity in sensory neurons.
- Through their life experiences, individuals learn the concept of pain.
- A person's report of an experience of pain should be respected.
- Although pain usually serves an adaptive role, it may adversely affect function and social and psychological well-being.
- Expressing pain can be done through various behaviors, not just verbal descriptions. That someone cannot communicate does not mean they are not experiencing pain, whether human or a non-human animal.

Pain is complex to study in humans as it can be interpreted as an emotional experience, the result of a sum of internal signals, an internal state, or many other ways.

Pain definition is a much-discussed topic [38], but it can be classified in multiple ways, depending on its origin, duration, and nature. The most commonly recognized types of pain are [39] :

1. **Neuropathic Pain**: Pain resulting from damage to or dysfunction of the nervous system, often described as burning, tingling, or shooting.
2. **Nociceptive Pain**: Pain that arises from potential or actual tissue damage being sensed by nociceptors, typically described as sharp, aching, or throbbing.
3. **Chronic Pain**: Persistent pain that lasts for 3 months or longer, often continuing even after the injury or illness that caused it has healed or gone away.
4. **Acute Pain**: A sudden and sharp pain that typically indicates injury and lasts a short duration, usually resolving as the injury heals.

5. **Radicular Pain**: Pain that radiates along the path of a nerve due to irritation or inflammation of the nerve root, commonly seen in conditions like sciatica.

Pain, as experienced by humans and animals, is a combination of one or multiple of these triggers, memory of past experience, and internal state. Pain can even be experienced without any of these triggers, resulting from only an emotional state.

Pain is often expressed inappropriately, for example, the Smoke Detector Principle (SDP) [40] posits that our body's protective systems, like pain or anxiety, often produce false alarms or exaggerated reactions because the minor inconvenience of these false alerts is outweighed by the potentially catastrophic consequences of missing a real threat, similar to how a smoke detector functions. The rubber hand illusions also highlight this SDP, demonstrating that pain can be experienced without any noxious signals being induced by the body.

Other dysfunctions of pain perception can be observed in patients suffering from chronic pain; pain is, in these specific cases, observed without evidence of tissue injuries. Some pain clinicians argue that chronic pain results from a mismatch of the pain system with the modern environment [41].

Predators seen or unseen, behaving in a threatening or non-threatening way; a potential mate or competitor for a mate, to be impressed or deterred by appearing healthy and strong [13]. This underscores that perceived pain can affect the impression of the environment.

Behavioral theories of pain emphasize how pain perception influences action-selection and behavioral responses. Skinner's operant conditioning theory suggests that pain-related behavior (e.g., rest-seeking or avoidance) is reinforced by external factors like attention or relief from responsibilities [42]. Pain leads to the prioritization of behaviors aimed at minimizing discomfort. Fordyce expanded this theory in chronic pain, proposing that persistent pain behaviors (e.g., limiting physical activity or avoiding specific tasks) are maintained by reinforcement even after the initial cause has been resolved. This highlights how pain can shift an individual's behavior, leading to avoidance patterns and altering action-selection in response to pain stimuli [43].

Some clinicians delved into the neurobiological foundations of pain, emphasizing pain is not only a sensory experience but an emotional experience and a cognitive phenomenon. In "Biologie des Passions," Jean-Didier Vincent [44] explains how pain is processed in the brain, specifically within the limbic system, and how complex interactions between neurotransmitters and hormones influence perception. These biopsychological perspectives highlight the predominant role of hormones like endorphins and cortisol in the regulation and perception of pain. This hormone can also play a more significant role in physiology and action-selection with mechanisms such as "gluconeogenesis," [45] where cortisol secretion leads to glucose generation mechanisms.

From a neuroscience perspective, pain is processed through different steps: transduction, transmission, modulation, perception, and emotional and behavioral response.

Pain perception results partly from noxious stimuli. Nociception is a biological process crucial for detecting potential or actual tissue damage [4]. It uses specific sensors, the nociceptors located in the skin, muscles, and organs, that detect harmful stimuli, triggering

signals through nerve fibers, including $A\delta$ fibers for sharp pain, to the brain. Research in robotics has explored nociception models, incorporating nociceptors in robot designs for simulating pain detection mechanisms [46, 16].

Alongside nociception, another sensory experience allows us to understand the physical interaction through touch. Specialized sensors related to both processes can be seen in Fig. 2.5. Mechanoception is the biological process through which the body perceives and interprets mechanical stimuli, including touch, pressure, and vibration [47]. This process is mediated by sensory cells called mechanoreceptors [48], which are distributed through the skin, muscles, and other tissues. These receptors respond to mechanical change, converting physical force into electrical signals transmitted through $A\beta$ fibers to the brain—specifically to the somatosensory cortex. The cortex processes and interprets the tactile sensation, enabling the construction of a physical body representation [49] (see, e.g., the *cortical homunculus* [50]).

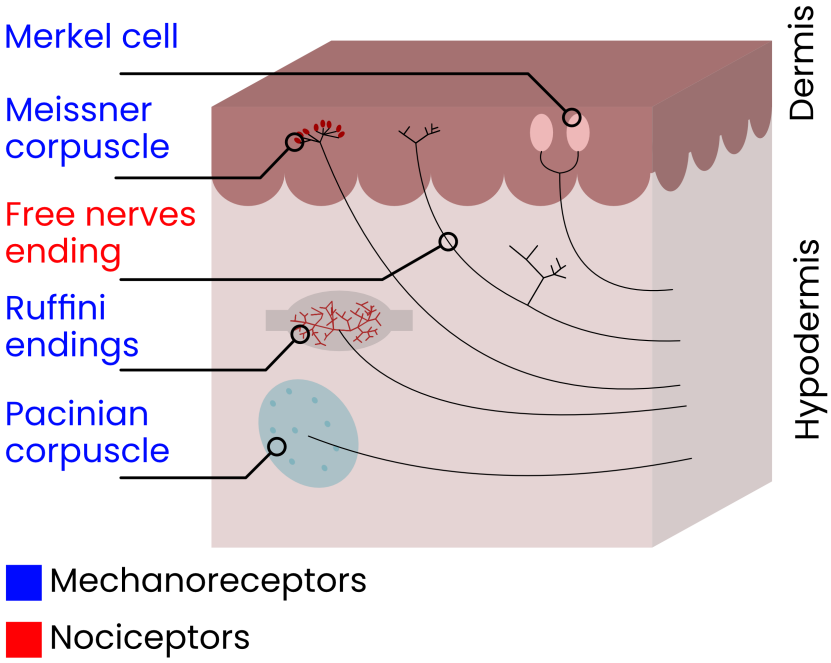


Figure 2.5: Shows four types of mechanoceptors and nociceptors involved in mechanoception and nociception biological process

This pathway leads signals from sensors located in the skin to specialized neurons in the body that send signals to the cortex via the spinal cord. Nociceptors and mechanoreceptors differ in signal transmission speeds; $A\beta$ fibers transmit touch and vibration quickly (30-70 m/s), while $A\delta$ fibers, associated with pain, conduct at 5-30 m/s [51]. This forms the basis for our neural network's computation of sensory outputs, leveraging the distinct speeds and activation levels of these fibers. As Melzack and Wall highlighted in the Gate Control Theory [52], the spinal cord plays a crucial role in controlling the flow of pain signals to the brain.

These signals are then relayed to different brain regions, such as the Anterior Cingulate Cortex, somatosensory cortex, or thalamus, where signals are processed and pain percep-

tion occurs. The somatosensory cortex is more specialized in the location of stimuli, while other regions, such as the Anterior Cingulate Cortex [53], specialize in pain perception.

Finally, these signals will be sent to the amygdala and hypothalamus to modulate the emotional and behavioral responses to pain perception.

Pain perception is a complex state that can thus be affected by the agent's internal state, memory, and perception of its environment. The challenge of its modeling involves a complex dynamic with many parameters.

Belkaid et al. explore how emotions influence the perception of peripersonal space (PPS) [54]. They suggest that emotional states, such as pleasure and pain, dynamically modulate how a robot perceives its surrounding space and behavior. This leads to an interesting question about embodied representation and how can pain and emotional states affect these.

Penfield and Rasmussen described the somatosensory homunculus in "The Cerebral Cortex of Man" [55, 56] as a map of the body's sensory input distribution across the cerebral cortex. It demonstrates how different body parts are represented in the brain, with larger cortical areas dedicated to more sensitive regions. Building on this, Hoffmann et al. [57, 58, 59] investigated how humanoid robots can form somatosensory-like representations of their artificial skin using local tactile simulation. They used self-organizing maps (SOM) [60], adding constraints on the maximum receptive field (MRF) size to mimic biological processes where the connectivity of neurons is localized to specific regions. They demonstrated the robot's artificial skin could form a representation that reflects the spatial arrangement of its sensors.

2.3 Computational and robotics models of pain

Over the years, affective computing and robotics have expanded significantly, exploring how artificial systems, including robots and agents, can simulate and respond to complex emotional and sensory stimuli, such as pain-related noxious ones, integrating insights from ethology, endocrinology, and neurosciences.

2.3.1 Sensory approach of pain perception models

Some researchers focused on the sensory process related to pain perception to model pain. This represents a complex challenge because inputs from the robot are often limited to proximity sensors like I.R., ultrasonic, or servomotor information. This information is far more limited than the complexity and nuance the skin provides, with texture, temperature, and pressure variation, for example.

Efforts have been made to develop sensors that mimic biological features. Yoon et al. [61] proposed an artificial nociceptor based on a diffusive memristor; they highlight the necessity of pain perception to minimize potential physical damage to robots. Parvizi-Fard et al. [62] also proposed an FPGA-based tactile neuromorphic system that could detect sharpness. This model adds potentially helpful information about what type of noxious stimuli robots face. Abdelwahed et al. [63] work on artificial skin may also offer a new

layer of sensory information that robotic agents could use to interact with environments in the future. With low cost and reproducible material, his approach offers a "dermis" capable of reconstructing the location of touch, pressure, and texture information. However, these approaches must still be compatible or applicable to current robotic systems. They remain theoretical and in the preliminary stages.

Maniscalo and Infantino [46] proposed an artificial model of nociception using Nao's robot servomotors' temperature and stress to model constraints on the robot body. This approach offers a solution for humanoid robots with several joint motors but does not fit mobile wheeled robots.

This highlights the difficulty of modeling accurate noxious stimuli for artificial agents. As more robots are built with limited sensors, we emphasized the need to reconstruct more complex information about sensory interaction using the robot's actual sensor to create an embodied robotic model with pain perception.

2.3.2 Embodied AI paradigm and Animats

Embodied AI is a paradigm in artificial intelligence which emphasizes the role of the body in cognition and intelligent behavior. This paradigm differs from traditional AI that focuses on abstract computations detached from physical forms. Embodied AI builds on the idea that intelligences emerges from the interaction between an agent's body and its environments. Rodney Brooks challenged the conventional symbolic AI approach with its subsumption architecture [64] showing how simple sensorimotor interactions can lead to complex behavioral response without relying on internal model or representations [65, 66, 11]. It has also been argued with the concept of "intelligence without representation" that cognitive processes are deeply rooted in the physical and situated nature of the agent, which interact with its surroundings [67]. We believe that physical interaction with the environments for pain perception modulation of action-selection.

The Animat approach (the contraction of artificial and animal) was inspired by Wilson [68], who settled the idea that an artificial agent, often a robot, is designed to simulate the behavior of living organisms, specifically animals. The idea is to model their ability to perceive and respond to environmental stimuli. This approach offers a reproducible and analyzable environment to assess the viability of models and theories that we could set up. Lewis [69] used a robotic agent to study OC-Spectrum disorders, underlying the value of animats for understanding human mental disorders. Animats help study homeostasis and action selection, and pain perception, like many other emotional states, can serve as a signal to modify behavior and restore balance.

2.3.3 Action selection and homeostasis

Action-selection, homeostasis, and motivation-based architectures are crucial to designing artificial agents capable of responding to environmental stimuli. Action-selection refers to the mechanisms by which an agent chooses from a set of potential behaviors to meet internal goals or respond to environmental stimuli such as threats. In biological organisms, this process is deeply connected to homeostasis, the principle by which organisms maintain a stable internal physiology. Damasio [70] emphasized the role of homeostasis

in the decision-making process, proposing that emotions serve as motivators for actions that preserve internal well-being. When homeostasis is disrupted (e.g., when hunger is experienced), the agent is motivated to take corrective action to restore internal equilibrium. Damasio [71] argued that there is not only physiological homeostasis but also an affect homeostasis. For him, homeostasis is the basis of biological and socio-cultural human life. This approach highlights the importance of internal balance physiologically and emotionally.

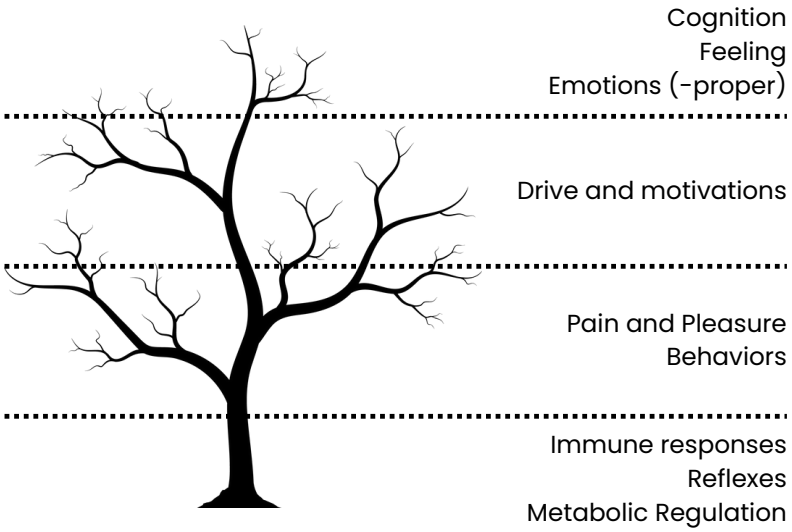


Figure 2.6: Damasio's representation of the levels of automated homeostatic regulation Adapted from [2].

We will embrace the principle of motivation-based architecture that draws inspiration from classic ethology and describes Damasio's [2] work on the different levels of automated homeostatic regulation (Fig. 2.6). This approach highlights the interplay between basic physiological processes and higher-level decision-making mechanisms. This process relies on a drive related to internal homeostasis that influences motivations that are the heart of action selection. Cañamero (1997) developed robotic action-selection mechanisms that build on this approach of motivation-based action selection. In this model, robotic agents must manage and choose between different behaviors to maintain their internal homeostasis in a dynamic environment with resources. This model relies on motivation-based architecture where different physiological variables represent the robot's internal states, and robots need to minimize errors in these physiological by selecting the correct behavior. This interaction between homeostasis, motivation, and action-selection is crucial for developing autonomous systems capable of adaptive responses to dynamic environments.

Affective computing is a field that focuses on the study and development of systems and devices that can interpret, process, and simulate human effects. It has been introduced by Rosalind Picard [72]. This involves the creation of artificial systems capable of detecting and responding to emotional and physical stimuli, which is both a sensory and emotional experience. Pain perception modeling presents a substantial challenge, as it requires

integrating multi-dimensional signal-sensory, emotion, and cognition into a framework that allows robots to react to pain-like stimuli in a way that mimics biological organisms.

Lola Cañamero proposed a motivation-based model of action selection [10] drawing inspiration on ethology where an agent had to evolve in a two-dimensional world and maintain its internal homeostasis with various physiological variables. To stay alive the autonomous agent has to select the appropriate behavior. Cañamero then proposed to use internal motivation which are in competition, linked to physiological deficit and perception of the environment. Later other works proposed to build on this architecture robotic models [69, 73, 74]. This model can also be used for active language learning [75]. We will embrace this approach of action-selection for our future work and build on these model.

2.3.4 Hormonal and emotional modulation

Krichmar [76] proposed that neuromodulatory systems in biological organisms (e.g., the mechanisms that regulate dopamine or serotonin) can serve as a framework to control autonomous agents. He emphasizes how neuromodulation enables organisms to switch between exploratory and exploitative behaviors based on environmental stimuli. This approach highlights how robots can better adapt to changing environments, improving action-selection mechanisms.

Teerakittikul's work investigates a novel Artificial Hormone Network architecture to enhance adaptability in autonomous robots for dynamic [77], unstructured environments, demonstrating that evolved hormone networks outperform manually designed systems in coping with internal and external changes.

Vallverdu et al. [78] have introduced a neuro-hormonal messaging architecture, which provides a conceptual roadmap for using hormonal bio-inspired models. This architecture merges neuronal signaling with hormonal messaging, which is crucial for understanding robotic emotional stimuli processing and response. The framework, drawing inspiration from the endocrine system's hormonal interactions, employs these signals as a means of communication between A.I. agents. This method mirrors human and animal hormone-influenced decision-making and behavior, as highlighted in the work of Avila-Garcia and Cañamero [79], who proposed hormonally modulated models for action-selection.

For social robots, Paiva Leite Ribeiro [80] provided an overview of the latest advances in emotion modeling for social robots, including the Affective Loop, explicit modeling of affective states, the use of Behavior Markup Language (BML) for multimodal generation, and the challenges of affect recognition, to enable social robots to interact with humans in ways that correspond to human emotions.

For pain perception analysis, hormonal computing provides a framework that starkly contrasts traditional deep learning models, offering a more interpretable and responsive system.

In the evolution of emotional models in robotics, the foundational Circumplex Model of Affect by Russell [1] initially helped understand the dimensional space of emotions, which is crucial for robotic emotion simulation. Vallverdu's advanced models build on this, integrating complex hormonal interactions and signaling mechanisms, marking a significant advancement in emotion modeling. Recent models have begun to incorporate frameworks

for pain perception, utilizing hormonal signals to modulate robotic behavior in response to pain stimuli. This development in understanding pain as a multi-dimensional construct includes the work of Feng and Zeng's [81] brain-inspired pain model based on a spiking neural network.

The work I will propose and discuss in the following chapters embrace this paradigm of hormonal emotional modulation as a framework to modulate action selection.

Chapter 3

Pain perception influence on an action selection model

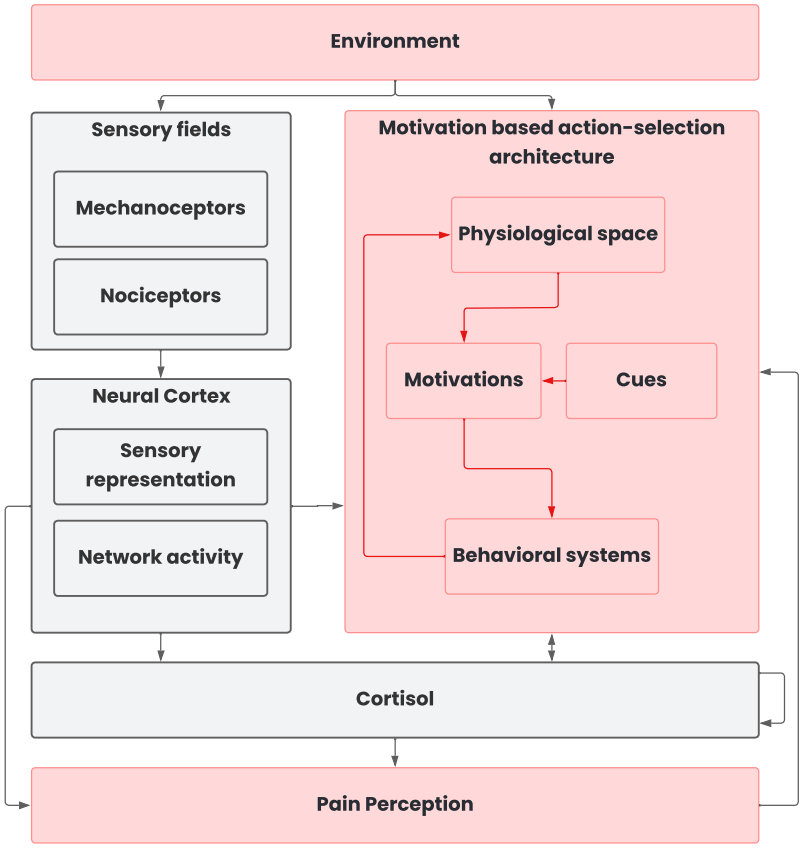


Figure 3.1: High-level diagram of our model and the effects we investigate. Red areas and arrows indicates the elements developed in this chapter.

In this chapter, I will first define our motivation-based action selection architecture. We will introduce a first model of nociception for damage perception, and then, we will introduce a pain perception model.

Pain is usually defined as an unpleasant sensory and emotional experience linked to actual or potential physical damage [4]. However, this definition is insufficient, and we should reassert the importance of pain as a subjective experience with social considerations, sensations, and emotions that are not only physiological[13].

Throughout evolution, this signal, which warns us about dangers to life, has given us advantages in environments with specific survival-related dangers, such as predators, by allowing us to perceive, respond to, and ultimately avoid such dangers. However, nowadays, environments generally have fewer survival-related dangers and present very different features to ancestral ones. In modern life, pain is less often than in ancestral times and is closely associated with survival threats or actual physical damage. Pain does not seem so closely related to survival-related threats (e.g., chronic pain), and the perception of pain does not necessarily have a strict correlation with physical damage—the pain signal might also be higher or lower than the actual level of damage. Since pain can be very disruptive and have negative consequences on people's daily activities, health, and well-being, it is not entirely clear whether it is still adaptive to feel pain [41].

In this chapter, I present a bio-inspired decision-making robot model and experiments to study the adaptive value of pain and its perception in environments with different amounts of survival-related dangers—ranging from high levels, as found in "ancestral" environments, to low levels, as in "modern and privileged" environments—and different levels of correlation between physical damage and pain. This model can help us understand pain perception and provide a useful "mirror" to understand how different sensitizations to pain can affect our behaviors. It can give a better understanding of the evolutionary origins of pain.

Building on prior work, I propose as an original contribution to draw inspiration from the biological features of pain and its sensory pathway, its behavioral impact and how it can modulate action selection. I will propose a first framework for damage perception with artificial nociceptors and use them to compute a pain artificial hormone.

3.1 Motivation based architecture

The evolution of behavior-based robotics has seen a significant shift from purely reactive agents driven solely by external stimuli to more complex models incorporating internal motivations. Rooted in ethnological studies, these advanced models emphasize the dual influence of internal and external factors on animal behavior and, by extension, robotic agents. Drawing insights from traditional animal behavior studies, motivation-based architectures emerged, highlighting the necessity to include internal states, such as essential variable levels, in the action selection process.

I propose a two-layer model —motivations and behaviors— linked by physiology architecture for our action selection model. This architecture build upon prior work on action selection by other researchers [12, 15]. Some elements may evolve but its core functioning and computational elements remains stable. However we will propose a new robotic platform to implement it and new features relative to noxious stimuli, pain perception and its general behavioral impact.

3.1.1 Physiology

The robots' physiology consists of several survival-related, thermostatically controlled variables. These variable defines a **physiological space**, or viability zone, within which survival is guaranteed. By crossing those boundaries, robot will "die".

Each physiological variable i is defined by a current value v_i, an ideal value θ_i and its deficit, or physiological error (Δ_i *or* $Phys.error$) which represents the difference between the ideal value and the current value and is computed as follows:

$$\Delta_i = \theta_i - v_i \tag{3.1}$$

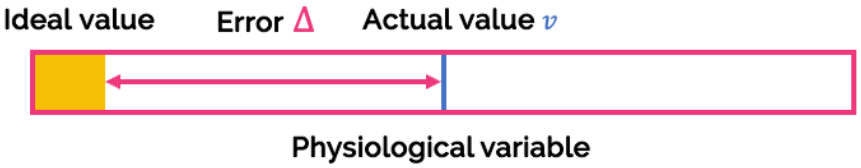


Figure 3.2: Representation of a physiological variable in which is defined ideal value, actual value, and error

The physiological space of a model is defined by N physiological variables that will represent a N-Dimensions space in which agent will need to maintain each of its component within a permitted range in order to survive.

The behavior actions modify the physiological space. A behavior can increase or decrease the level of a physiological variable. To maintain homeostasis and keep physiological variable values within the permitted range, the robot needs to execute specific behaviors that will reduce the error. Other behaviors will increase the physiological error.

3.1.2 Sensors (internal and external)

The specific robot I use for this model is a Thymio (Fig. 3.3), an Aseba robot designed for education and some robotic and artificial intelligence research. Thymio is a small compact square with one rounded-edge robotic platform designed for education and research in robotics and artificial intelligence. It has a circular shape and is 5.3 cm in height and 11 cm by 11.2 cm in length.

I use the following Thymio **external sensors** to detect relevant elements of the environment and to create **internal sensors** relevant to specific tasks of the model:

- **Proximity Sensors**: InfraRed (IR) sensors located in the front and the back of the body of the robot are used to detect objects up to 10cm from the robot. For convenience, I will talk about "Proximity sensors." IR sensors provide values between 1023 (object next to the robot's body) and 0 (object beyond the sensor's range). Proximity sensors are normalized to values in the range 0 to 1.

- **Ambient Light Sensors**: The IR sensors underneath the robot are used to detect the two different types of resources situated on the floor of the arena.
- **Resources sensors**: for our specific action selection problem, the robot needs to consume resources to maintain its internal physiological variable within the permitted range. Objects of a specific color represent resources. Thus, I use ground ambient light sensors to compute **internal sensors**, which will represent the detection of such resources.

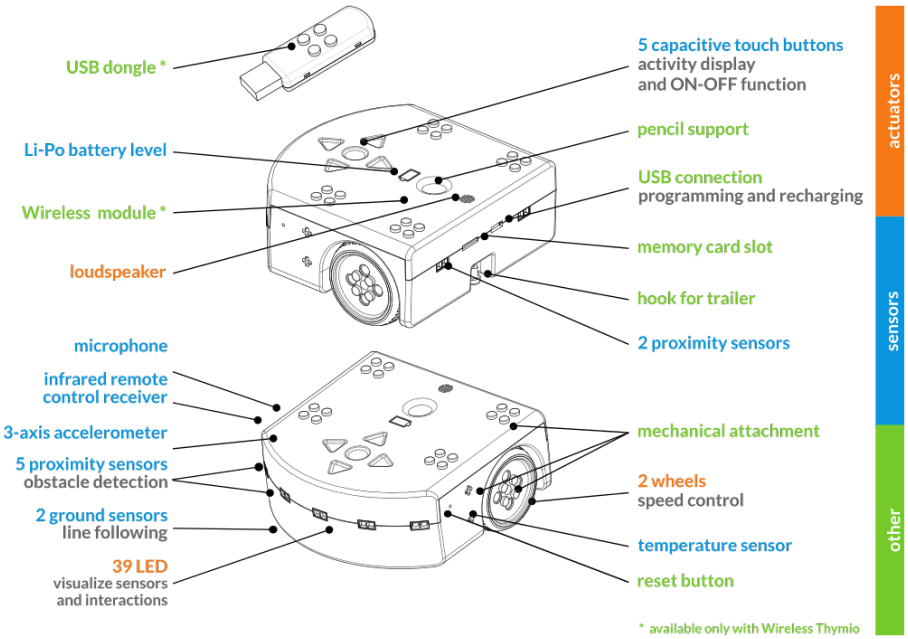


Figure 3.3: Thymio specifications as given by robot manufacturer [3]

3.1.3 Incentive cues (external stimuli)

Action selection is influenced by the presence of external stimuli (i.e. incentives cues) that will influence the robot's perception of the environment. Cues can be related to noxious signals and gives useful information about robot trajectory (avoidance of obstacles) or about the seek-and-consume behaviors (does the robot perceive resources ? Can it consume resources?).

3.1.4 Motivations

Motivations for the robot represent a will to act. They signify the tendency to want to act in a certain way based on external and internal factors. The internal factors can be errors in physiological spaces, called "drives", which urge the robot to maintain the state of physiological variables by reducing their error and thus, keeping the values within the allowed viability ranges. The external factors are incentives cues that allow the perception of the environment.

For each physiological variable, I compute a related motivation based on an ethology formula [82] as follows:

$$motivations = phys.error + (Phys.error * cue)$$

However, I also decided to implement a specific motivation related to obstacle avoidance. I consider physical integrity to be a specific internal state the robots need to maintain by avoiding obstacles, which can be represented by lowering the incentive cue related to obstacle perception.

Thus, I get motivation intensity at each time, which allows us to select the behavior that will best fit the robot's internal state and execute appropriate actions in the environment. I use a **Winner-Takes-All** (WTA) strategy where the motivation with the highest intensity at each time will select the linked behavior as seen in Fig. 3.4.

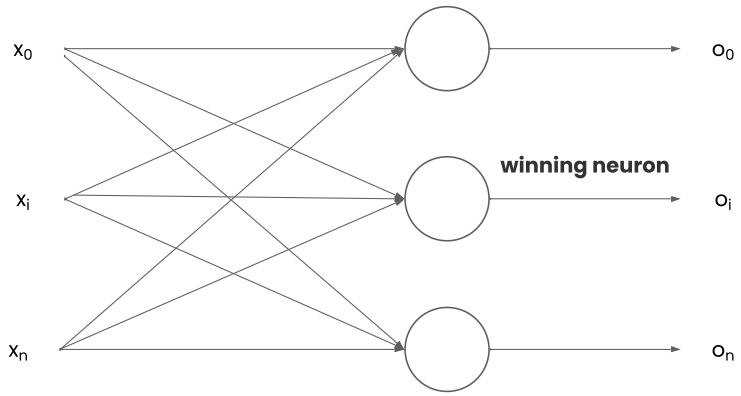


Figure 3.4: Winner Takes All competitive selection. x represents one of the n inputs, linked to each neurons and to each one of the o outputs. the ith greater input activate the ith neuron which activate the itth o output of the network, selecting the greater input.

Behavioral systems

Behavior systems (BS) are collections of behaviors that are related to motivations.

Our robot architecture is comprised of the following Behavioral Systems (BS), which include both "consummatory" (goal-achieving) and "appetitive" (goal-seeking) behaviors.

- **Seek & Consume** is a complex behavioral system composed of simpler behaviors, both "consummatory" (goal-achieving) and "appetitive" (goal-seeking) [83], organized in "layers". This architecture draws inspiration from Brook's subsumption architecture [11]. The layers are: consume resources - seek for resources - wander around the environment. The BS will try to execute the "higher" (consummatory) layer first and, if this is not possible, will try the lower ones as described in Fig. 3.5. Seeking behavior is implemented using a Braitenberg [84] model, see Eq. 3.3.
- **Reactive**: a behavioral system that provides the robot with a "danger escape" behavior. It only contains one sub-behavior (implemented using a Braitenberg model, see Eq. 3.2) to run away from obstacles.

$$\begin{pmatrix} motor_1 \\ motor_2 \end{pmatrix} = \begin{pmatrix} -3 & -2 & -1 & 2 & 3 \\ 3 & 2 & -1 & 2 & 3 \end{pmatrix} \cdot \begin{pmatrix} IR_{left} \\ IR_{frontleft} \\ IR_{front} \\ IR_{frontright} \\ IR_{right} \end{pmatrix} \tag{3.2}$$

$$\begin{pmatrix} motor_1 \\ motor_2 \end{pmatrix} = \begin{pmatrix} -2 & 2 \\ 2 & -2 \end{pmatrix} \cdot \begin{pmatrix} IR_{ground_{left}} \\ IR_{ground_{right}} \end{pmatrix} \tag{3.3}$$

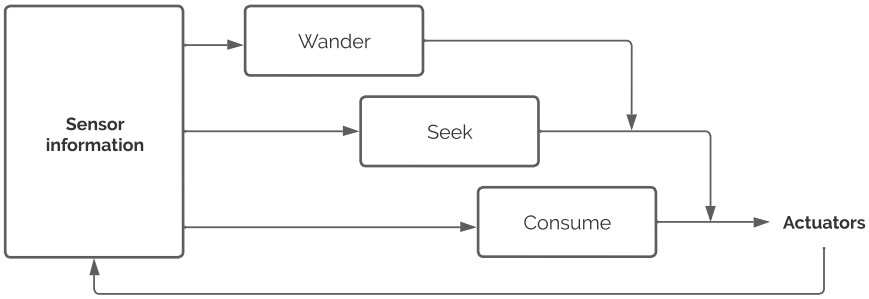


Figure 3.5: Seek & Consume subsumption inspired behavioral system.

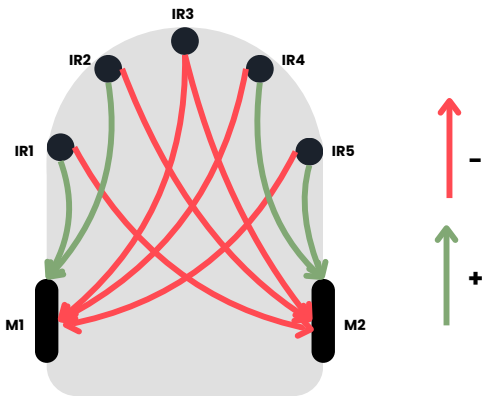


Figure 3.6: Braitenberg vehicle used for obstacle avoidance, red arrows indicate negative weights, green arrows positive weights

Each behavioral system is designed to satisfy a motivation, and each sub-behavior affects the relevant robot's physiological variables, increasing or decreasing their levels.

3.1.5 Activity cycles

Activity cycle represents the evolution within the physiological space due to action selection. As can be seen in Fig. 3.7 internal physiological variables can be represented within

a n dimensional space, n being the number of physiological values for a robot. In this space a balanced homeostasis area can be defined by representing constraint boundaries. If one or multiple physiological deficits become too important related to the others, internal homeostasis becomes unbalanced and survival is threatened. We can observe in this space the impact and effect of action selection selected behaviors by observing how a deficit can decrease or increase, and thus observe if our model correctly maintain the internal physiological balance.

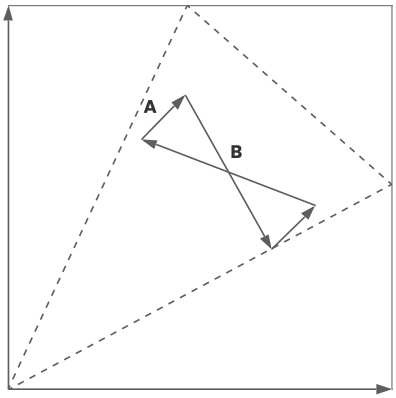


Figure 3.7: Activity cycles, graphical representation of physiological space error. Dotted line represents the safe space within which survival is guaranteed and homeostasis is balanced. Arrows represents evolution of deficits over time. (A) represents the increase of an error for one physiological space resolved in (B) by the consumption of a resource.

3.2 Nociception & pain perception

Pain is [38] an unpleasant sensory and emotional experience associated with actual or potential tissued damage, thus it is important to see pain not as a displeasure but as a result of a noxious stimuli. Nociception is the sensory process related to noxious stimuli and is the result of the specialized sensors in skin called Nociceptors [85]. I propose a new way to model the way a robot perceives damage with artificial nociception. This artificial nociceptive process will lead to a damage perception that we will compute using an artificial hormone that will simulate the pain perception of the robot [86]. Pain has many biological features that may be interesting to modulate action selection that we will investigate such as the "second pain principle" or "damage irradiation" [4].

3.2.1 Artificial Nociceptors

The first model of artificial nociceptors I propose draws upon inspiration from biology and intends to offer the primary perception of the sensorial environment of the robot. We will consider our robot surface as an **artificial skin** composed of **artificial nociceptors**

[46]. We will use the IR sensors to create them and detect whether or not the robot is "touched". Inspired by mechanoceptive nociception, we will implement two types of nociceptors sensitive to different noxious stimuli.

Impact nociceptors

Our first type of nociceptors is based on the *speed of the impact*; thus, I will use our *IR sensors* to compute the speed of approach of the object and, following a linear line, the quicker the obstacle goes on, the stronger the object is engaged, the more the nociceptor is excited.

$$speed = \frac{\Delta d}{T_{iteration}} \tag{3.4}$$

I compute this speed for each sensor and then store it for each iteration. If speed increases from iteration to iteration, the nociceptor signal will increase proportionally.

Scratching nociceptors

I propose a second type of nociceptor sensitive to "scratching" movement represented by an object moving quickly from one sensor to another (Fig. 3.8).

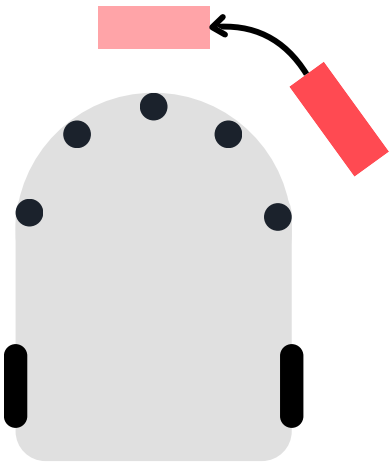


Figure 3.8: Graphic representation of a scratching noxious stimuli

To determine a nociceptive signal with this kind of movement, we must first compute the angular speed of the object moving following the following equation where θ is equal to a fraction of 2π corresponding to the degree separating our robot sensors ($\theta = \frac{\pi}{6}$ and $r = 5,5cm$ for Thymio-II).

$$speed = \frac{\theta r}{T_{iteration}} \tag{3.5}$$

Speed can be computed by generating an array of *IR* sensor values and a second array stacking previous iterations' sensor values. Then, we can compare previous values with close neighbor sensors and use a previous formula to get the speed of the moving object.

We repeat this and doubly increase damage for each iteration if an object keeps scratching more and more sensors.

Nociceptors & Damage irradiation

For each IR sensors we get two nociceptors sensible to our two noxious stimuli, impact and scratching. We compute for each of our 5 IR sensor a "general nociceptor" which combines the two different types of noxious stimuli.

$$nociceptor_i = \frac{impact_i + scratching_i}{2} \tag{3.6}$$

Where i stands for the ith nociceptor sensor.

Pain irradiation principle [4] highlights that noxious stimuli in a location will generate a wider perceived damage area with the unpleasant sensory radiating to close areas of the initial noxious contact zone. I propose to implement this *damage irradiation* using a Gaussian that propagates intensity to each nociceptor's neighbors:

Algorithm 1 Damage radiation algorithm

1: **Generate 5 arrays of 5 values** $array_i[5]$
2: **for** $i = 1$ to 5 **do**
3: $\quad array_i[i] \leftarrow nociceptor[i]$
4: $\quad$ **Following a Gaussian distribution:** Intensity of $array_i[i]$ will radiate to its neighbors
5: **end for**
6: **for** $i = 1$ to 5 **do**
7: $\quad nociceptor[i] \leftarrow \frac{\sum_{j=1}^{5} array_j[i]}{5}$
8: **end for**

Once we get our final nociceptors' value, we compute a general "damage" based on the mean of all the nociceptors' values.

3.2.2 Pain

I proposed a nociceptive process to quantify and compute the noxious stimuli, resulting in a "damage" perception. I propose to compute the pain perception of the robot as an "artificial hormone" based on this damage computation.

Hormonal computation

Pain perception is the result of the artificial nociceptive sensory process, I propose to compute it as an "artificial hormone" release by a *gland* related to damage.

Glands release hormones over time, depending on specific stimuli. I propose to compute the release rate r_{pain} of our artificial pain hormone correlated with pain.

$$r_{pain} = \alpha * damage \tag{3.7}$$

α is a significant coefficient that allows us to determine if the pain usually is **correlated** to damage or if it is **hypo-correlated** or **hyper-correlated** to damage. α gets three different states, representing the three different correlation conditions between damage and pain.

With this release rate, we compute a hormonal concentration c_{pain}. I proposed three ways to compute this concentration level:

- $c_{pain}(t+1) = min(1, c_{pain}(t) * \psi_{pain} + r_{pain})$ where there is a memory of hormone with ψ_pain is the decrease rate.
- $c_{pain}(t+1) = (r_{pain} + c_{pain}(t)) * 0.5$ where there is a short time memory with a mean approach of the concentration
- $c_{pain}(t+1) = r_{pain}$ where there is no memory

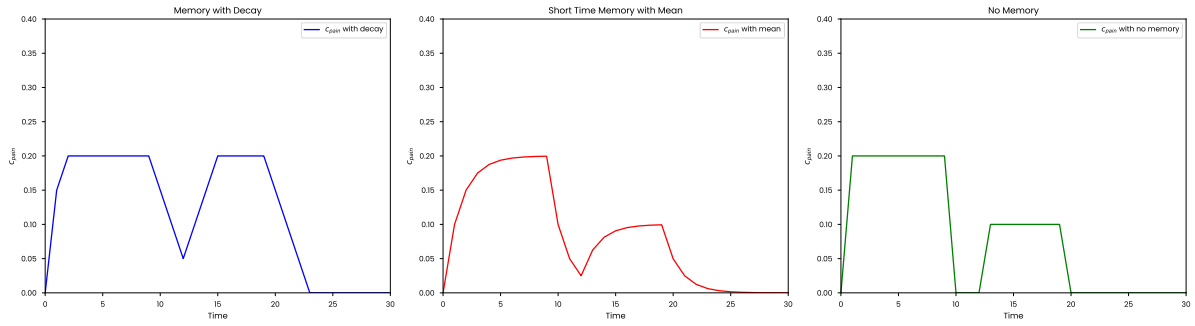


Figure 3.9: Visual representation fo the three types of hormonal concentration I propose. Blue is with decay rate, red with short term memory and green with no memory. For all three methods, a stimuli was induced during a fixed time then followed by a second less important stimuli.

I propose to use decrease rate method to compute hormonal concentration in our model because it keeps concentration memory longer than other models and allow to get back to higher concentration when to close stimuli are experienced as observed in Fig. 3.9.

Principle of "second pain."

We saw with pain irradiation that noxious stimuli radiates in an area, the second pain principle [4] highlights that pain is not only experienced in a specific time but it perception evolves over time. After a noxious stimuli, the pain perception will evolve over time with two different peaks of unpleasant sensory. A first intense pain will be followed by a second lower burning heating experience. We can summarize this idea over two peaks of pain, one higher and tighter followed by a lower but wider second peak. Thus, I propose to vary the pain perception over time once the pain concentration is computed, using a bimodal distribution (Fig. 3.10) in order to generate the two peaks, then we will take the maximum value between the value c_{pain} and the distribution *secondpain* over time depending on c_{pain}.

$$secondpain(x) = \max\left(A_1 \cdot e^{-(B_1(x-C_1))^2}, A_2 \cdot e^{-(B_2(x-C_2))^2}\right) \tag{3.8}$$

Where A_1 and A_2 are respectively the **amplitudes** of the first and second peaks, B_1 and B_2 controls the **widths** of each peak, and C_1 and C_2 **shift the position** of each peaks along the time axis.

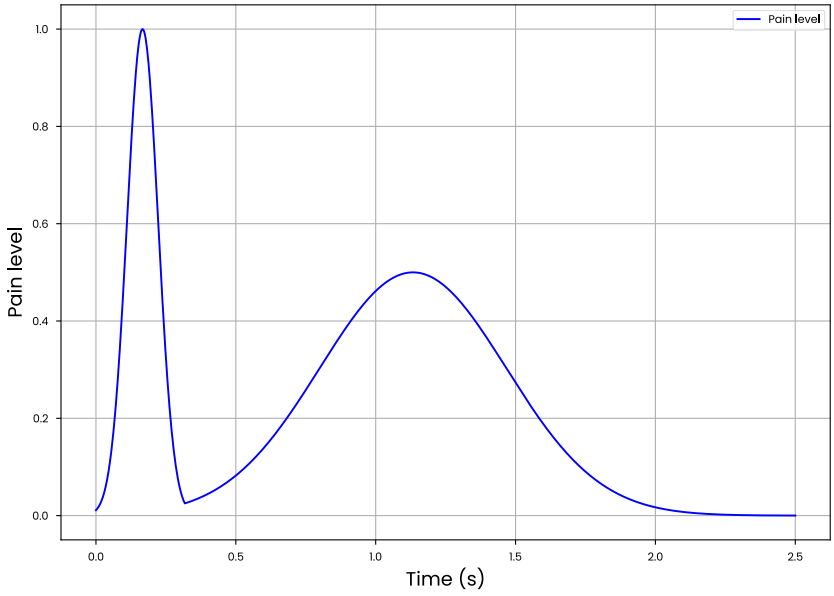


Figure 3.10: Bimodal distribution used for second pain computation

3.2.3 Pleasure

For our robot model, I will also compute a **pleasure hormone** based on **well-being**, inspired by Cos Canamero [87], to fit the hedonic pleasure of resource consumption.

$$wellbeing = 1 - (\Delta_{energy} + \Delta_{integument} + \Delta_{integrity}) \tag{3.9}$$

Where Δ stands for the deficit of a physiological space.

Then, we compute the release gate and hormonal concentration using the same principle we used for pain perception:

$$r_{pleasure} = wellbeing \tag{3.10}$$

$$c_{pleasure}(t+1) = min(1, c_{pleasure}(t) * \psi_{pleasure} + r_{pleasure}) \tag{3.11}$$

3.2.4 Impact of pain & pleasure on the action selection model

Pain has a strong impact on our behavioral response to noxious stimuli. Drawing inspiration from Amanda Williams's work [13] we will propose a twofold impact on both the motor command and the motivation to act. These features intend to improve the survival in danger-related danger environments and we will observe how our model is impacted by this modulation.

First, I propose to modify the motor command of the robot to fit the idea that *"pain make engaging its actions strongly"*. We will modify the modulate the strength of action by reinforcing the actual speed with a factor modulated by the pain perception.

$$speed_{l/r} = speed_{l/r} + \alpha \cdot (1 + c_{pain}) \cdot sign(speed_{l/r}) \tag{3.12}$$

Second, we propose to use *"escape feature"* adaptive value of pain to reinforce the obstacle avoidance motivation.

$$m_{avoid} = m_{avoid} + m_{avoid} * \beta * c_{pain} \tag{3.13}$$

On the other hand, as we consider pleasure as the general wellbeing related to the hedonic satisfaction of consuming resources, and so-on, reducing the deficits of physiological spaces, we propose pleasure to have an impact on food and grooming motivation by adding a bonus over time:

$$m_i = m_i + \beta * c_{pleasure} * m_i \tag{3.14}$$

3.2.5 Motivation-based action selection model

Building on all these elements, we can graphically summarize our model in Fig. 3.11. We also summarize our model's different physiological elements and internal variables in Table 3.1 and Table 3.2.5.

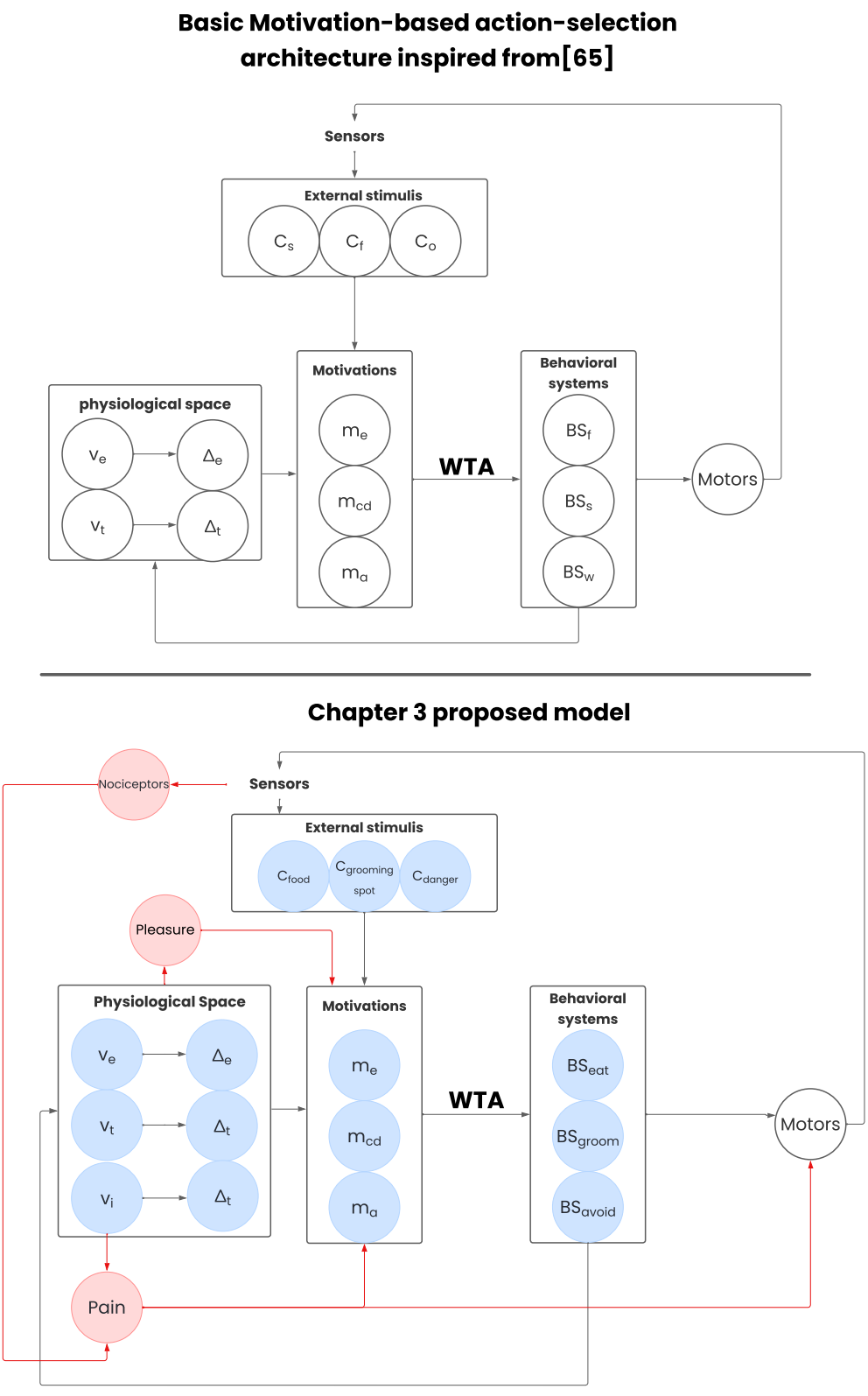


Figure 3.11: Granular view of the motivation-based action selection robot's model compared to the one we draw inspiration on. Red elements and arrows are the new elements we added and blue elements are the specific elements modified without touching computation and logic

Elements	Name
Physiological variables (v)	Tegument, temperature, integrity
Sensors (internal & external)	Energy, Grooming Spot, Ground IR Sensors, Nociceptors
External stimuli, or cues (c)	"food," "grooming spot," "danger"
Motivations (m)	hunger, groom, danger
Behavioral systems	Eating behavior, Grooming behavior, Avoiding behavior

Table 3.1: Elements of the action selection architecture.

At each iteration, energy and tegument decreases, when damage is induced, integrity is reduced.

Behavioral System	linked motivation	Behavior	Effects
Eating behavior	hunger	consume food	↑ energy
		Seek food	
		wander	
Grooming behavior	groom	Groom	↑ tegument
		seek for grooming spots	
		wander	
Avoiding behavior	danger	Avoid obstacles	

Table 3.2: Different behavioral systems, their linked motivation, behaviors and effects. ↑ indicates that the behavior increase the level of a physiological variable, ↓ that it decrease it level.

3.3 Experiments and results

3.3.1 Experimental setup

We establish an experimental environment where the robot can evolve and consume the two types of resources that must satisfy the physiological needs of our robot. For this, we create a physical arena with, on the ground, tiles of different colors (*Gray*: for a neutral zone, *Black*: for a grooming area, and *White*: for a food resource). Each tile will be A4 (21.0 by 29.7 cm). The arena consists of 16 tiles that lead to a 59.4 by 84.1 cm arena. The arena (Fig. 3.12) is materialized with a wooden wall to prevent the robot from escaping.

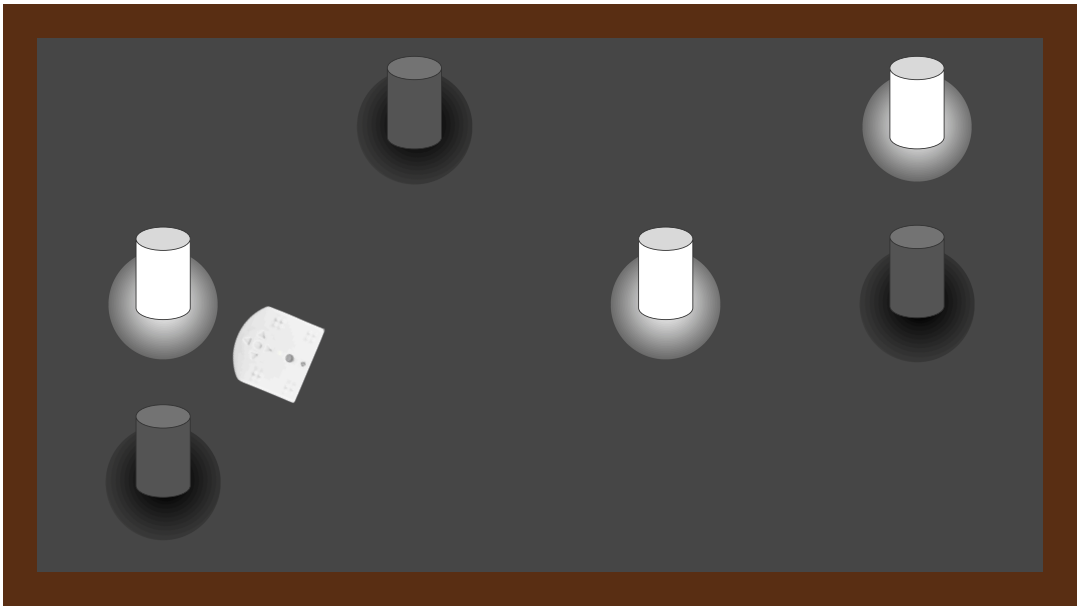


Figure 3.12: Graphical view of Thymio-II evolving in its environment with grooming spots (black tiles and obstacles) and food resources (white tiles and obstacles).

In order to carry out the experiments, the following protocol was developed:

- The arena was generated with random tile placement at the beginning of each run
- The robot was placed in the bottom middle of the arena, pointing to the top
- The robot was launched, when it "died", the experiment was over

All along the run, we recorded the Lifespan, physiological variables, hormones, motivation intensity, and selected behavior.

We designed three scenarios presenting different levels of survival-related danger to study the adaptive value of pain. We varied the presence of predatory attacks and obstacles in the arena. Each scenario was run five times with hyper-hypo, an average correlation between damage and pain, and no pain as a comparison group.

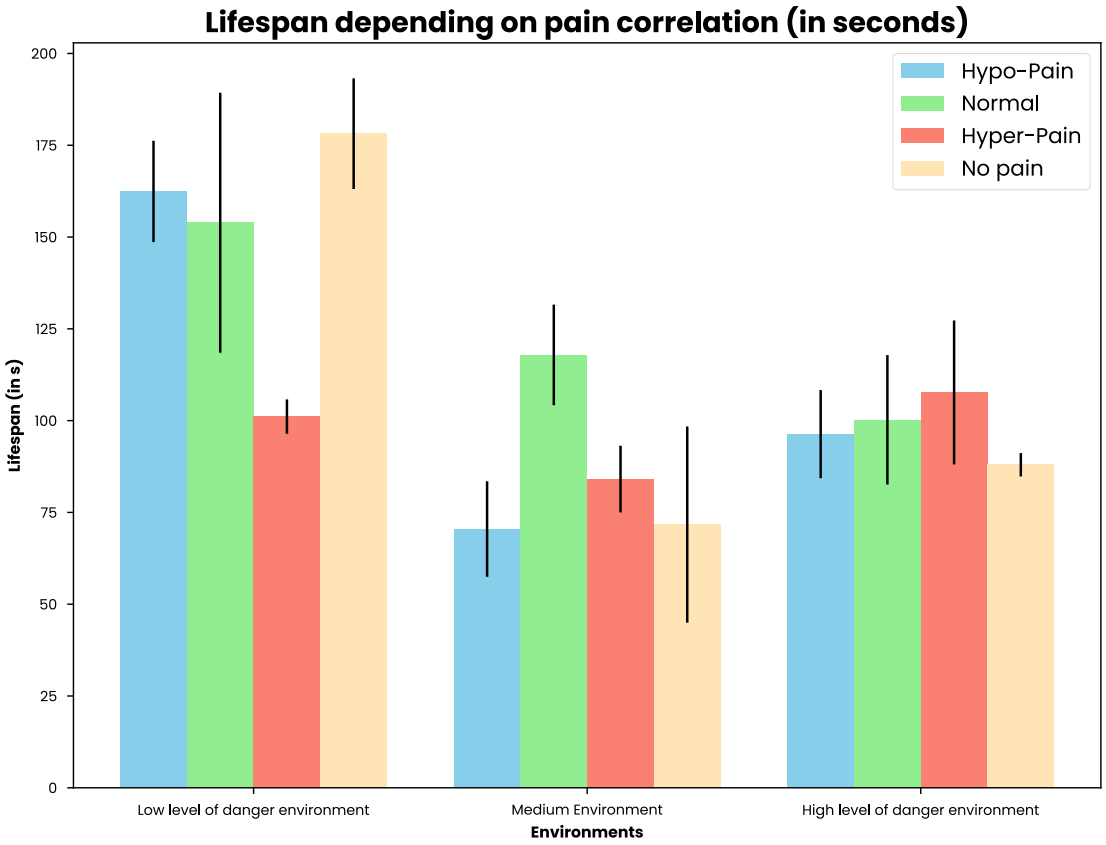
- *Scenario 1:* no obstacles and no predators
- *Scenario 2:* obstacles and no predators
- *Scenario 3:* obstacles and predators

Obstacles on the grooming spots and food resources add a dual value to the resources [69], as consuming them can lead to damage-induced pain perception. Thus, our action selection model will need to choose between actually consuming the resource or fleeing the danger.

3.3.2 Results in terms of lifetime

As observed in Fig. 3.13 there are notable differences in *lifespan*:

- *For scenario 1:* we can see Lifespan is lower when there is pain. The more pain experienced, the shorter the lifespan. Hyper-correlation between damage and pain leads to under-opportunism behaviors, where robots try so hard to escape possible danger that they do not consume resources. Regarding standard derivation, hyper-experience of pain leads to certain death in less time. Regular and hypo-correlation can induce the same Lifespan as the absence of pain but less certainty
- *For scenario 2:* we see pain is adaptive if we look at Lifespan when pain is experienced less or firmly with hyper and hypo correlation between damage and pain, adaptive value is lower. Looking at the standard deviation, we see that hypo-correlation is more reliable than the absence of pain, although there is no significant difference in Lifespan.
- *For scenario 3:* We see that the more pain is experienced, the greater the Lifespan is. Pain hypersensitivity is remarkably adaptive in this scenario, which may support the hypothesis that pain is adaptive in scenarios reproducing prey-predator mechanisms and inhospitable environments. We can see that experiencing pain makes the experience more diverse regarding Lifespan with more significant standard derivation.



	Hyposensitivity to pain	Strict Correlation	Hypersensitivity to pain	No pain
Low level of danger environment	162.43	153.9	101.1	178.16
Medium Environment	70.47	117.9	84.08	71.7
High level of danger environment	96.32	100.22	107.68	87.98

Figure 3.13: Lifespan (in s) depending on pain-damage correlation and scenarios

3.3.3 Results in terms of Causes of Death

Another interesting data to look at to understand our model and assess the adaptive value of pain are the Cause of Death (*Cod*) of the robot (Fig. 3.3.3):

- For scenario 1, we observe that the robot dies mainly from lack of energy but not from lack of Integrity.
- For scenario 2, we see that the robot can die for lack of physical Integrity. However, this does not happen with the presence of pain, regardless of its correlation with the physical damage.
- For scenario 3, we observe that the robot dies more due to its physical Integrity. It is the leading Cause of death when damage is hyper-correlated with pain. The robot will consume the resources within its reach more efficiently, even if it hurts.

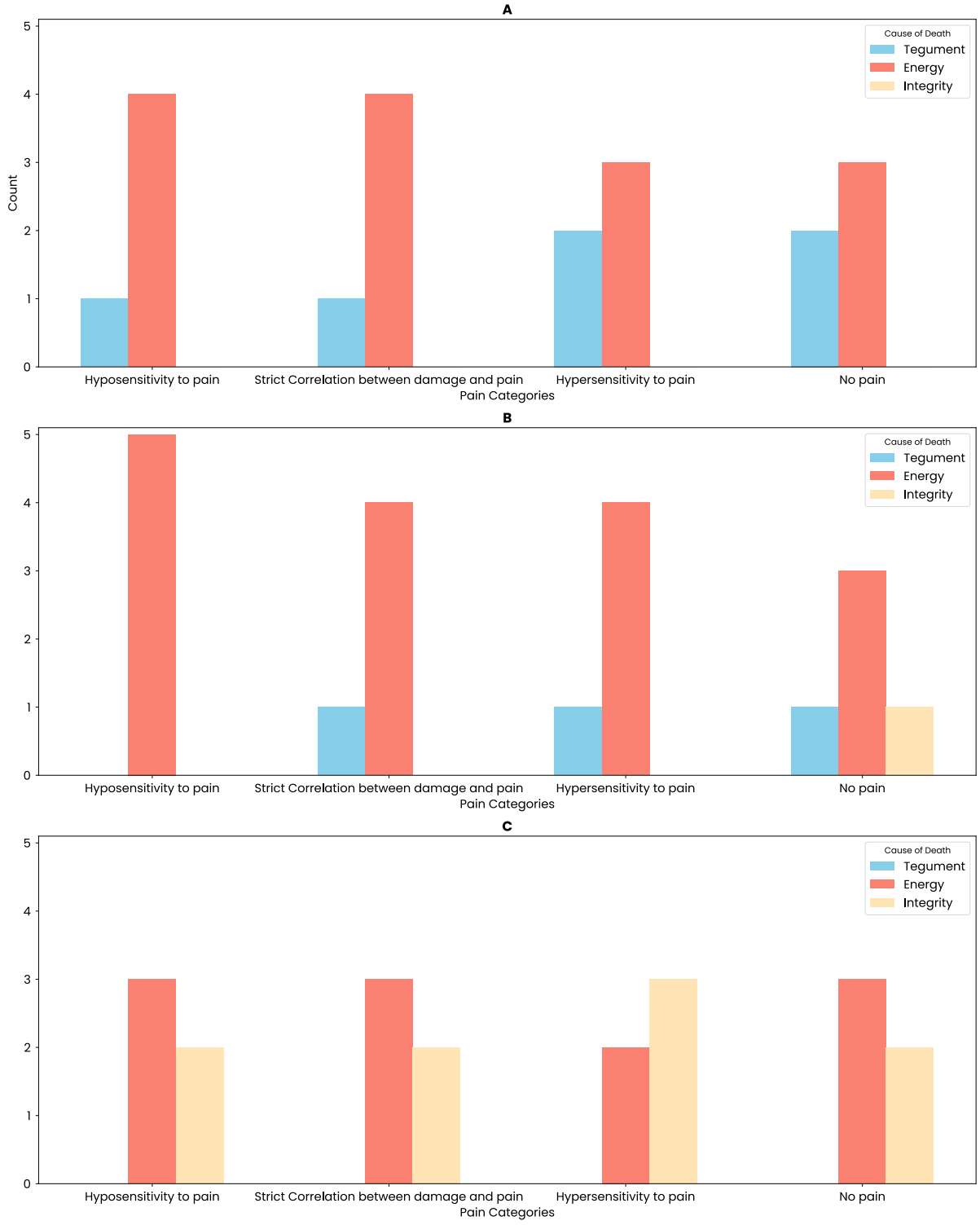


Figure 3.14: Cause of Death (*CoD*) for scenario with (A) low level of danger, (B) Medium level of danger, (C) High level of danger. Blue is death due to Tegument, Red to Energy, and blue to integrity

3.3.4 Some Notable runs

To study the other metrics we recorded, we detail some prototypical runs of the robot according to the scenarios and the correlation between physical damage and pain to understand our model and the impact of pain on it.

Hyper-correlation between pain and damage in scenario 3 (Fig. 3.15). In this scenario, pain seems adaptive regarding *lifespan* and *CoD*. We can observe that pain rises to a high peak quickly and remains at a high level with long peaks. This leads to avoidance behaviors and resource consumption when the robot is close to resources. We observe a lot of resource consumption, even when the pain is high, which confirms that the robot will consume resources, even if it attacks its Integrity. In this run, the robot dies for lack of Integrity;

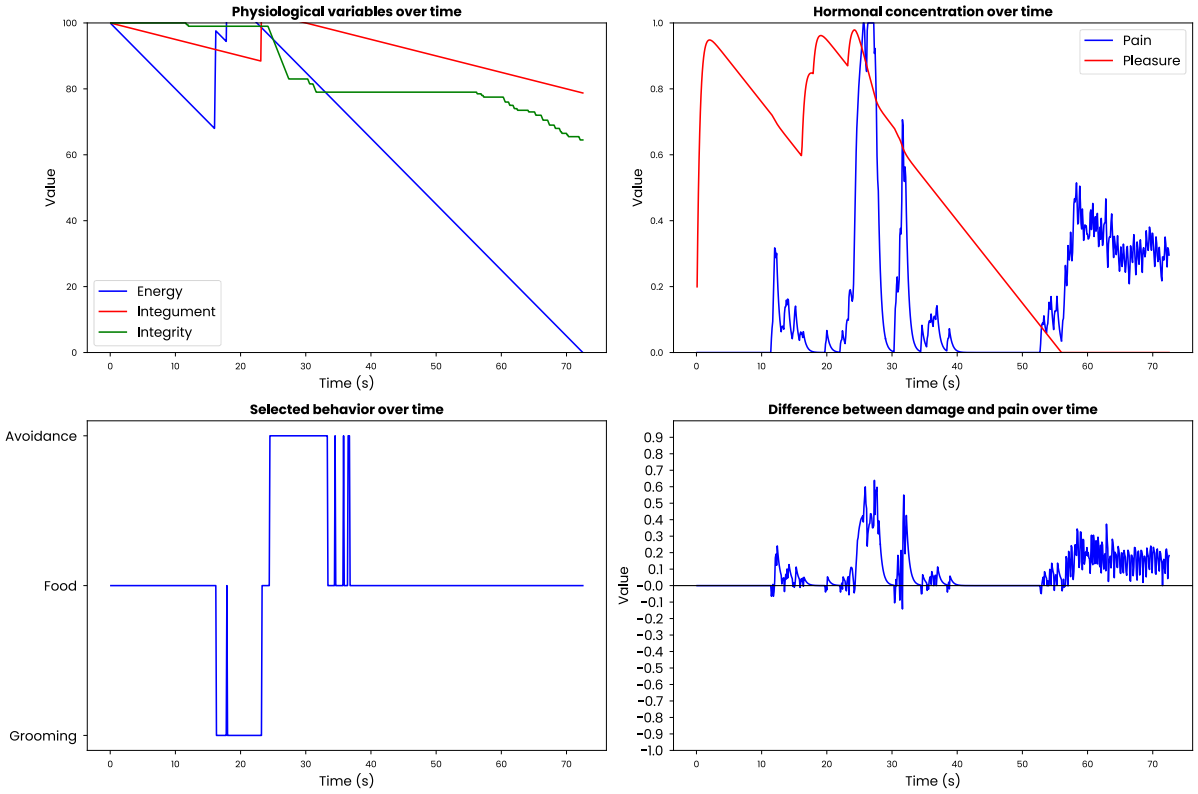


Figure 3.15: Metrics for Hyper-correlation between pain and damage in scenario 3. Metrics are as follows: physiological variables, hormonal concentration, selected behavior, and difference between damage and pain over time.

No pain in scenario 3 (Fig. 3.16). If we compare it to the previous run, we can observe how the model reacts differently in the absence of pain. We observe that the robot will consume fewer resources. We also see that even with high peaks of damage, robots still search for resources yet avoid consuming them when they are found.

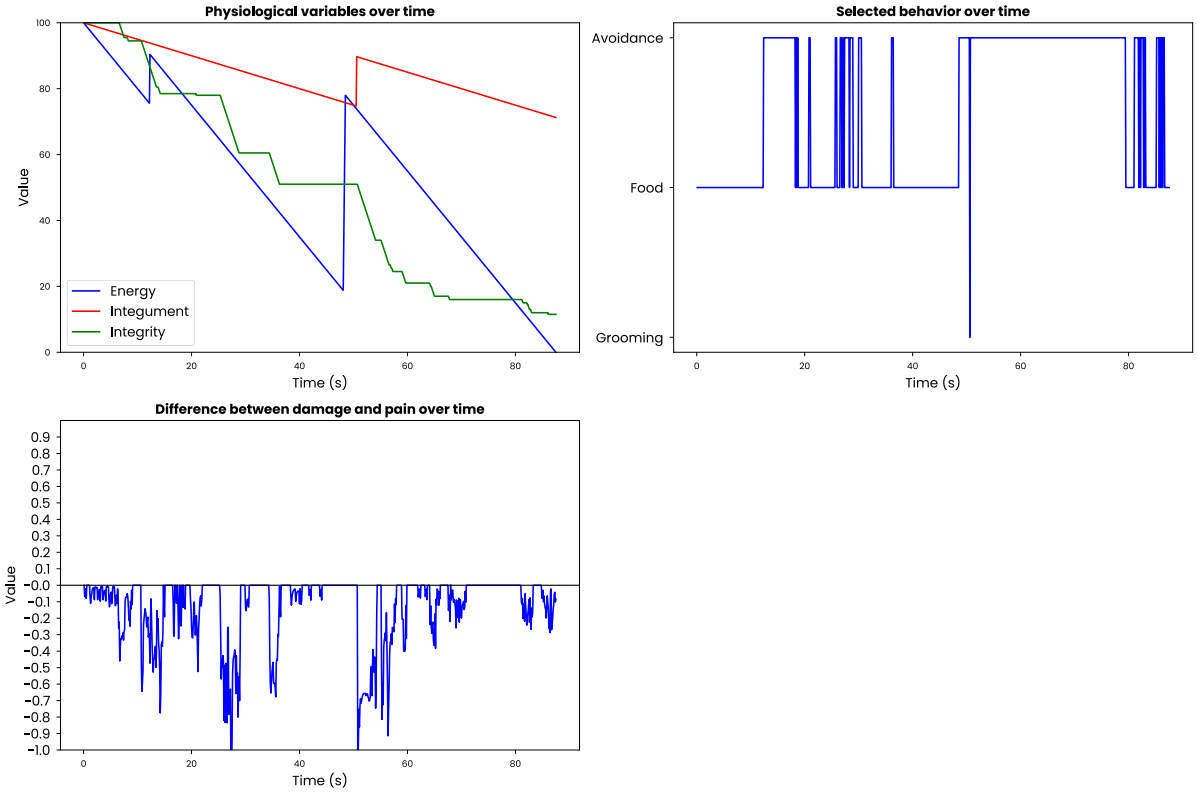


Figure 3.16: Metrics for No pain in Scenario 3. Metrics are as follows: physiological variables, hormonal concentration, selected behavior, and difference between damage and pain over time

Normal pain in scenario 2 (Fig. 3.17). This run is interesting in understanding the adaptive value of pain and its impact on the model. We observe a lot of resource consumption and more stable physiological values than on previous runs. However, there are still compulsive eating behaviors, even when unnecessary. Death is caused by modulation by pain, which leads to preferring obstacle avoidance over resource consumption at the end of life.

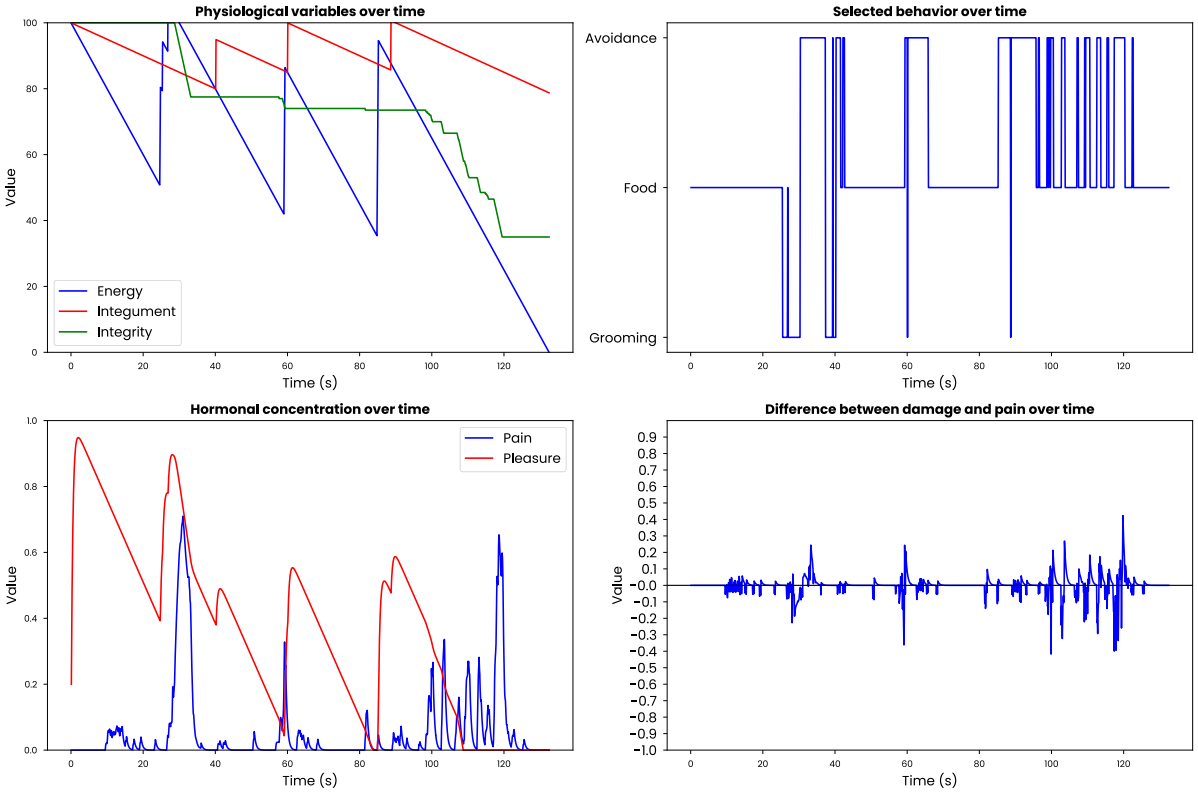


Figure 3.17: Metrics for Normal pain in scenario 2. Metrics are as follows: physiological variables, hormonal concentration, selected behavior, and difference between damage and pain over time

Hypo-correlation between pain and damage in scenario 1 (Fig. 3.18). This run is interesting because it is an *"extreme case"* to be compared to hyper-pain in scenario 3. We see that physiological levels are maintained at a high level until a significant decrease in energy is caused by the selection of an avoiding obstacle behavior. This happens later than in other scenarios yet still leads later to under-consumption, which explains the longer survival.

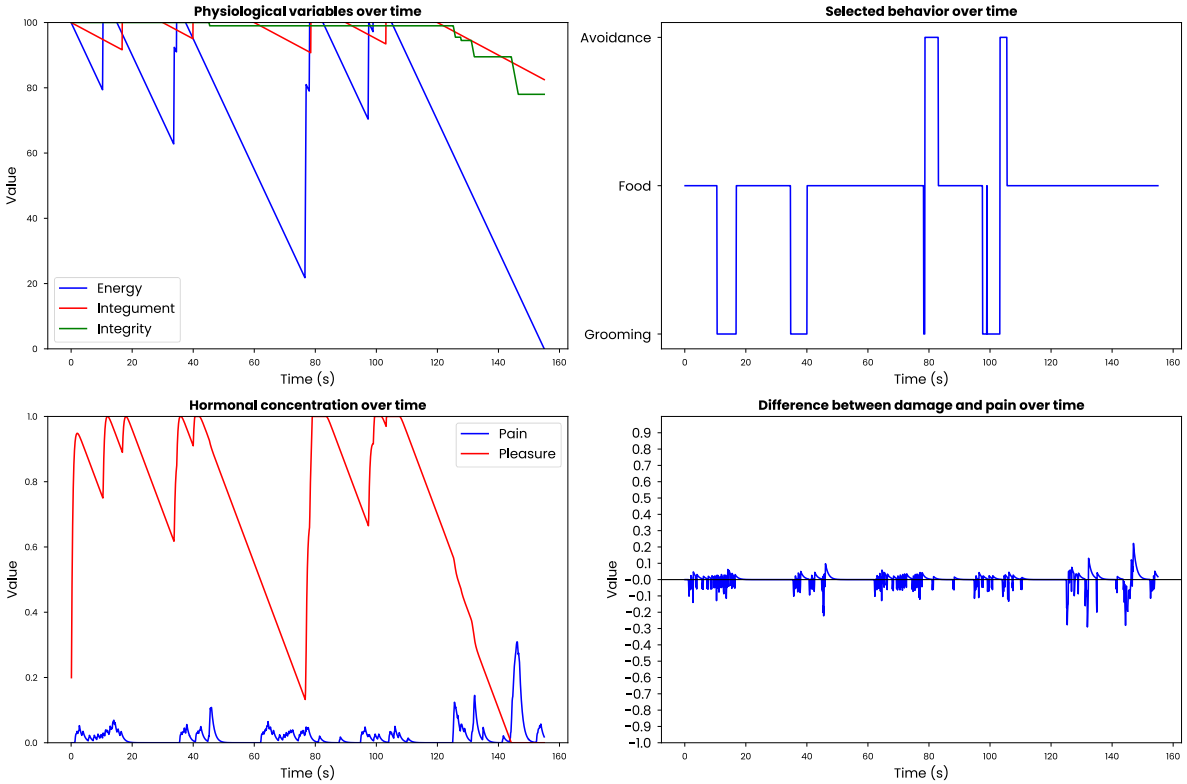


Figure 3.18: Metrics for Hypo-correlation between pain and damage in scenario 1. Metrics are as follows: physiological variables, hormonal concentration, selected behavior, and difference between damage and pain over time

3.4 Comparison with another model of nociceptors

We compare our model with another work from Maniscalo and Infantino [46], who proposed a pain model for a humanoid robot based on a form of artificial nociceptors. Their model uses "soft sensors" or "virtual sensors" to determine the pain level. They consider two types of pain induction:

- The first is based on the stress of the motors. When the movement of the robot is blocked, or someone blocks this movement, the motors are in a stress condition, and this induces pain
- The second is on the temperature of the robot; above a level, pain is induced

Based on these two criteria and using the following formula (Fig. 3.19) where *inhi* and *mod* respectively represent the inhibition and modulation of actions:

$$P_{c/t}(t) = \begin{cases} P_{c/t} = inhi * \text{mod} * \left(1 - e^{-t/\tau_c}\right) & if \quad C(t) > C_{Pain} \\ P_{c/t} = inhi * \text{mod} * e^{-t/\tau_c} & if \quad C(t) \leq C_{Pain} \end{cases} \tag{3.15}$$

To compare models, we replace pain computation in our model with their equations. We consider that circular and speed damage computations are used as stimuli to replace

motor stress and temperature. We then reproduce the experiment in *scenario 2* with hypo, hyper, and normal correlation between damage and pain. The following results are obtained after five runs of each condition;

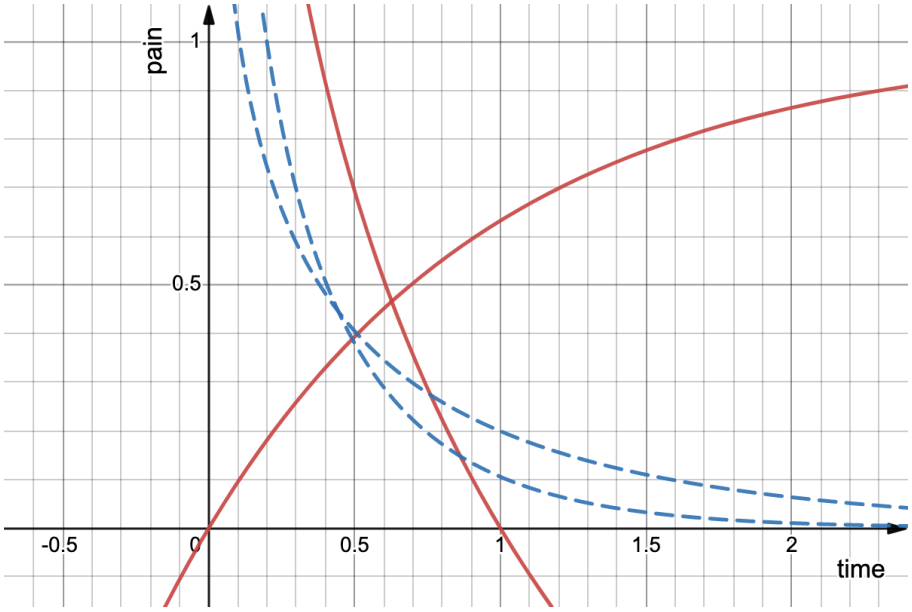
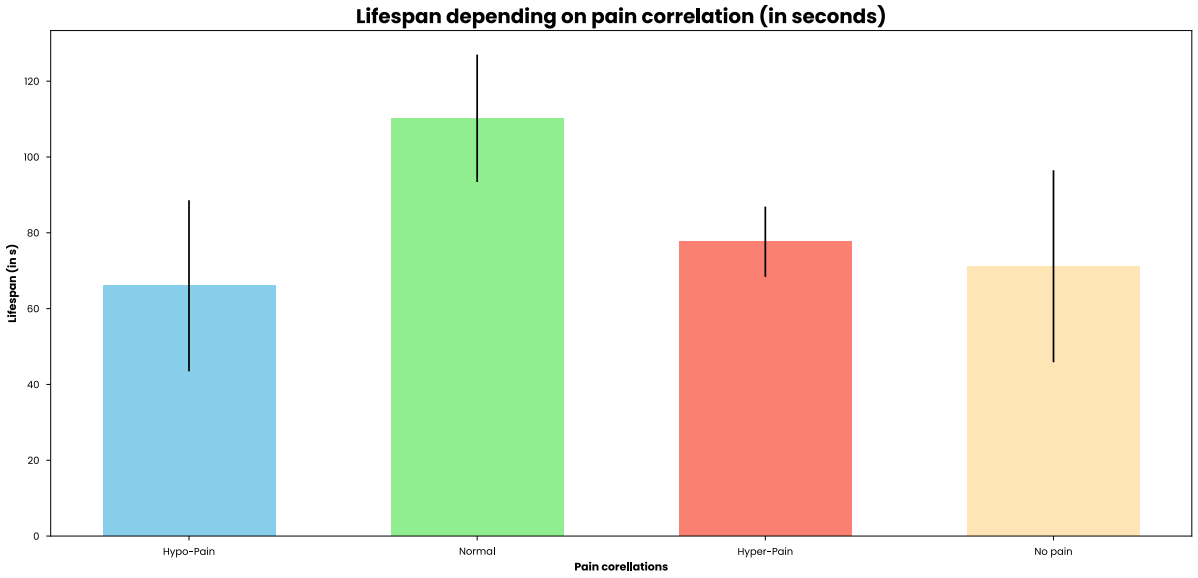


Figure 3.19: Graphic representation of pain induction, red is pain level, blue is pain discharge



	Hypo-Pain	Normal	Hyper-Pain	No pain
Moy	66.02	110.2	77.64	71.18

Figure 3.20: Robot's Lifespan (in s) depending on pain-damage correlation

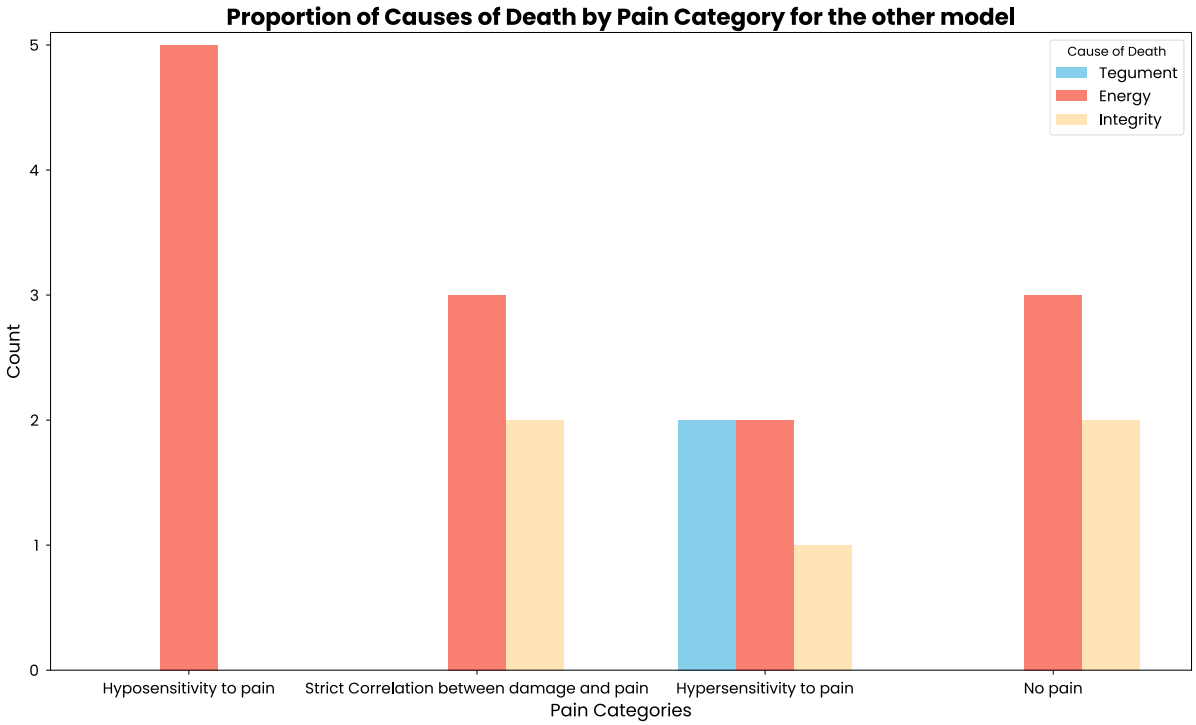


Figure 3.21: Cause of Death (*CoD*) for second model. Blue is death due to Tegument, Red to Energy, and blue to integrity

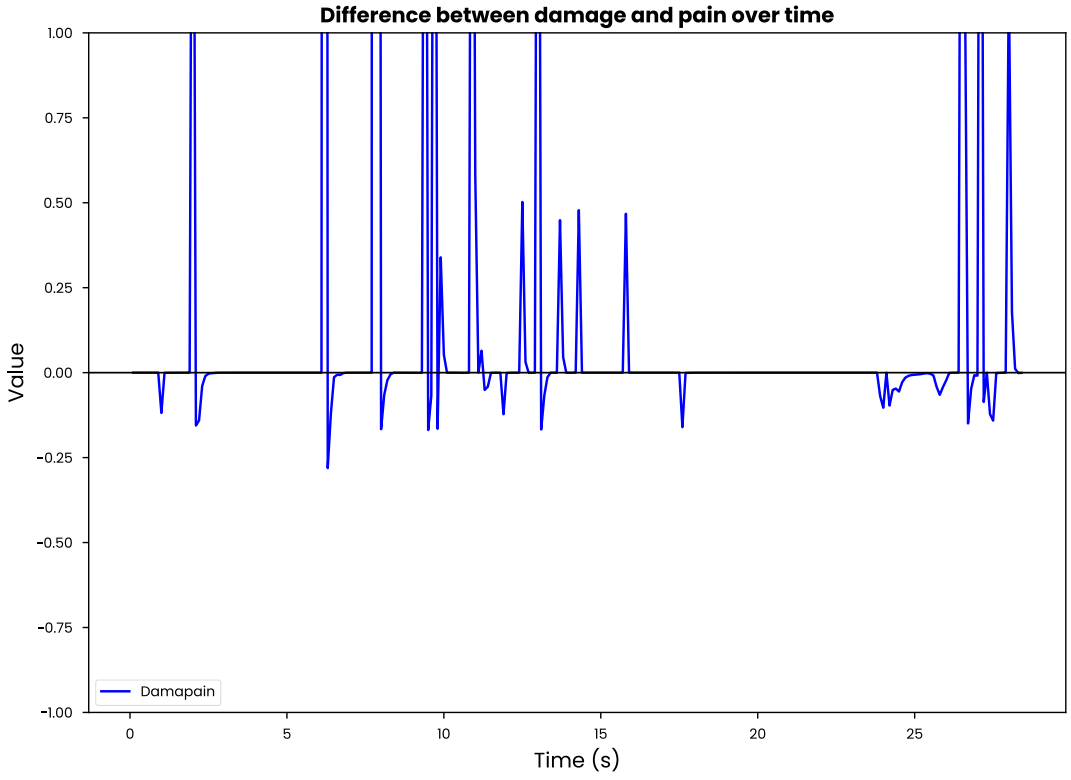


Figure 3.22: Difference between damage and pain over time.

We observe in Fig. 3.20 that, regardless of the correlation between damage and pain, Lifespan appears similar, yet slightly lower, than with our model.

Their model seems less efficient in responding to our problem because all Lifespan appears lower.

In the causes of death (Fig. 3.21), we observe that robot tends to die more often because of its integrity. If we look at the correlation between damage and pain when pain is usually correlated with damage (Fig. 3.22), we observe that pain is present in higher and more intense peaks when the damage reaches a certain level, which is caused by the *threshold effect* with C_{pain} and the exponential side of the pain computation, which makes it saturate quickly.

By comparing these results to those of our model, the two models seem practical in the context for which we have developed our model, despite their different approaches to the relationship to pain.

Maniscalco and Infantino's model offers a more direct view and a more specific study of the relationship between nociceptor-derived stimuli and pain. It directly focuses on the direct correlation between damage and pain, which is usually related to quick and short pain stimuli. Our model proposed an approach more linked to pain perception rather than a damage model.

3.5 Discussion

In this chapter, building on a motivation-based action selection model inspired by previous works, I proposed an embodied model of pain perception for a robotic agent and the nociceptive sensory process for detecting harmful and noxious stimuli, and thus, damage.

I reconstructed interesting characteristics of pain perception we find in biological pain, both in terms of spatialization with pain irradiation and in terms of temporal dynamics with the second pain principle. These allow the robot to have a more complex and closer to biology embodied perception of the noxious stimuli it is exposed to.

I proposed an artificial hormone model to simulate this pain perception directly linked to the sensory perception of damage, constructed from the artificial nociceptors I developed. This pain perception impacts both the agent's motivations to act and its motor actions, enabling the robot to respond differently depending on its emotional state.

We conducted a series of experiments in various environments containing different levels of danger, which reflect ancestral environments with many threats, notably predatory ones, and more modern environments with lower levels of danger and no predation. We also varied the relationship between damage and pain perception. By proposing these two variables, we were able to modulate both the "danger" in the environments and the way the robot perceives signals related to this danger.

We studied both the Cause of Death (COD) and the lifespan of our agent in these different environments and with different pain perceptions. The results indicate that hyperacuity to pain is adaptive in extreme, hazardous environments with high levels of danger, while insensitivity to pain is more advantageous in environments with less danger.

Walters and Williams proposed that chronic pain results from a mismatch between modern environments and our pain system [88]. Our findings tend to support this theory by highlighting the highly adaptive nature of pain in dynamic environments, where an agent needs to escape predators by both modulating its motivations to act and responding more strongly, engaging more intensively in motor actions necessary for survival, whether fleeing a predator or consuming resources more quickly to maintain internal homeostasis, and thus survival.

In our environment, consuming resources too quickly or forcefully could activate the artificial nociceptors, which tended to limit the advantage offered by pain and allowed for a more neutral analysis of its impact. Indeed, even in ancestral environments, experiencing an excessively strong perception of pain leads to a trade-off where the robot must choose between fleeing and consuming while risking injury. Even with this characteristic, it appeared from observing the different metrics of our model in various scenarios and CODs that hyperacuity to pain remained advantageous in an extreme environment. However, in a moderate environment, neither insensitivity nor hyperacuity was more beneficial than a "normal" pain perception based on a strict correlation between nociceptive damage and pain perception.

Finally, we compared our nociceptor model to that of Maniscalco and Infantino [46], which is based on computational neuroscience. We assumed a different approach on our way to compute and to give embodiment to pain perception with hormonal concentration that leads to memory of past experience. We also provide a more adaptive framework that focus less on the direct relationship between a noxious stimuli and the perceived damage but more on this idea of pain as an emotional state for the robot that modulate its behavioral response to the stimuli, leading to more embodiment.

Chapter 4

Hormonal (cortisol) modulation & temporal influence of pain perception on action selection

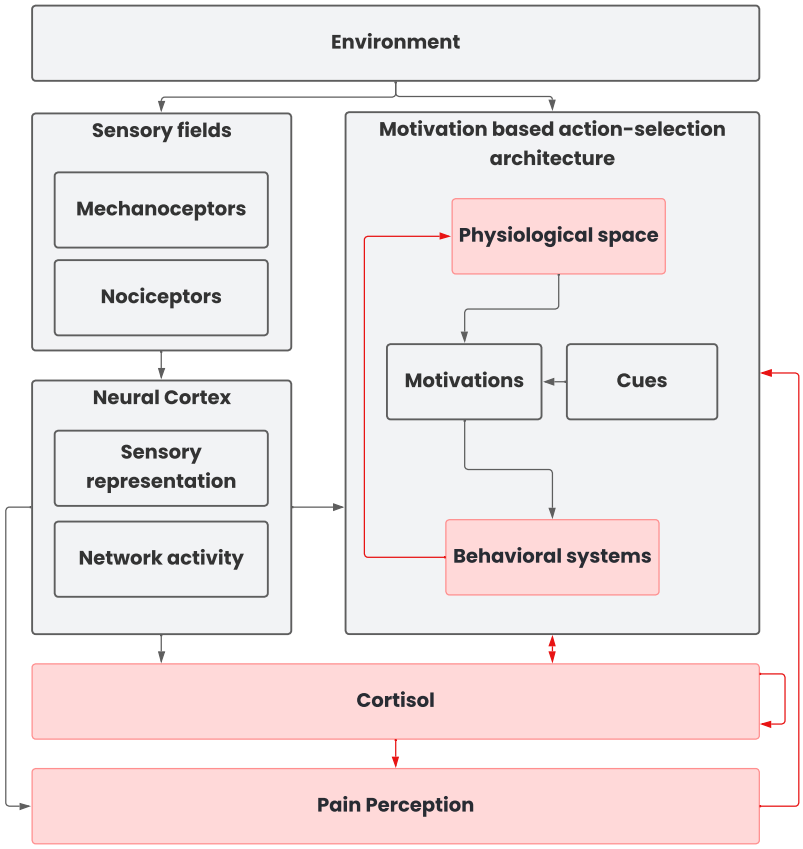


Figure 4.1: High-level diagram of our model and the effects we investigate. Red areas and arrows indicates the elements developed in this chapter.

Action selection is crucial for the survival and success of both biological entities and artificial agents, such as robots. These agents, which can be either computer simulations

or robotic systems designed to emulate certain behaviors or cognitive functions found in animals, play a vital role in the chapter and application of complex adaptive systems, learning algorithms, and the implementation of artificial intelligence. One significant challenge these agents encounter involves deciding on actions that ensure the maintenance of internal stability, or homeostasis, by acquiring necessary resources. This resource-seeking behavior can expose them to risks, leading to stress, damage, or metaphorical pain. According to evolutionary perspectives [89], one of the primary sources of stress and pain is the threat posed by predators in the environment.

As already mentioned in chapter 3, pain is an important feature of our survival system. It is a complex sensory state that integrates many sensory details; it conveys an unpleasant sensory and emotional experience linked to real or potential physical damage [4, 13]. It is a perceptual and affective experience [90]. Its perception is influenced by emotional state and environmental contingencies. Allowing escape mechanisms or attention-grabbing, pain is a survival-related mechanism and has an important influence on the mechanisms of action selection. In addition to the nociceptive noxious signal, the perception of pain is influenced by various factors [89], including the concentration of cortisol—often related to the hormone of stress [91]—in the bloodstream [86].

Nociception is the process through which the nervous system perceives and interprets harmful stimuli [92, 93], such as tissue harm or inflammation. In response to stress, the adrenal gland produces cortisol [94, 95]. The timing of pain perception influences how agents react to stressful situations over time. For instance, hysteresis, where a system's response is influenced by both its current and past states, can cause a delayed reaction to environmental changes.

In this chapter, I build new features in the previous action selection architecture we saw in Chapter 3 and test it in various ecologically inspired environments. These environments present challenges akin to those faced by animals in the wild, allowing us to explore how robots, as agents in resource-seeking scenarios, navigate and adapt to similar survival challenges. I aim to study the complex interplay between stress, pain and resource seeking, and how these factors can affects agents in different environments with varying level of predation and temporal differences in exposure to predation.

Employing robots to study this interplay serves two significant purposes. Firstly, it enhances our understanding of pain perception through controlled, systematic experiments that are not feasible with animals in natural settings. This is achieved by precisely altering experimental variables. Secondly, it advances the field of robotics by progressing towards the development of robots that are more adept at sensing and reacting to potential dangers in their surroundings.

This chapter extends the work previously published by L'Haridon and Cañamero on the role of simulated cortisol in Wellbeing and the adaptive value of pain [16].

I will investigate the impact of stress on pain perception in the context of action selection, where an agent, a robot, must seek and consume resources and avoid dangers in order to survive in a stressful environment. We will examine how different environments with different levels of predation-related danger and different temporal experiments can affect the robot's ability to select correct action and perceive pain. We will measure its lifespan and the management of its internal parameters. By observing and investigating these factors, we aim to provide insight into how agents can adapt and improve their ac-

tion selection mechanisms and resource-seeking behaviors in stressful environments and improve their chances of survival.

The question we want to answer is how a simulated hormone such as cortisol can improve the action selection model in a pain perception-modulated robot model. Our hypothesis is that simulated cortisol will improve the viability of the model, and they will, by the temporal mechanisms that define them, lead to habituation or sensitization to pain.

In the specific experiment we conducted, we could observe some results that tend to confirm our specific hypothesis, and we also observed some emerging behaviors and so on, characteristics of our model.

4.1 Action selection architecture

The specific robot model and action selection architecture that we have implemented (Fig. 4.2) are inspired by previous work on homeostatically-controlled motivation-based robot decision-making (behavior selection) architectures confronted to a *Two-Resource Problem* (TRP) [74], and the effects of stress on decision making [95, 96], but on this occasion applied to how stress can modulate the perception of pain, and how this combination can affect decision making and viability management. In this TRP, a Khepera IV robot [97] needs to maintain its two internal "physiological" variables within a viable range of values that ensures its survival, by seeking and consuming resources available in the environment.

4.1.1 Specific architecture

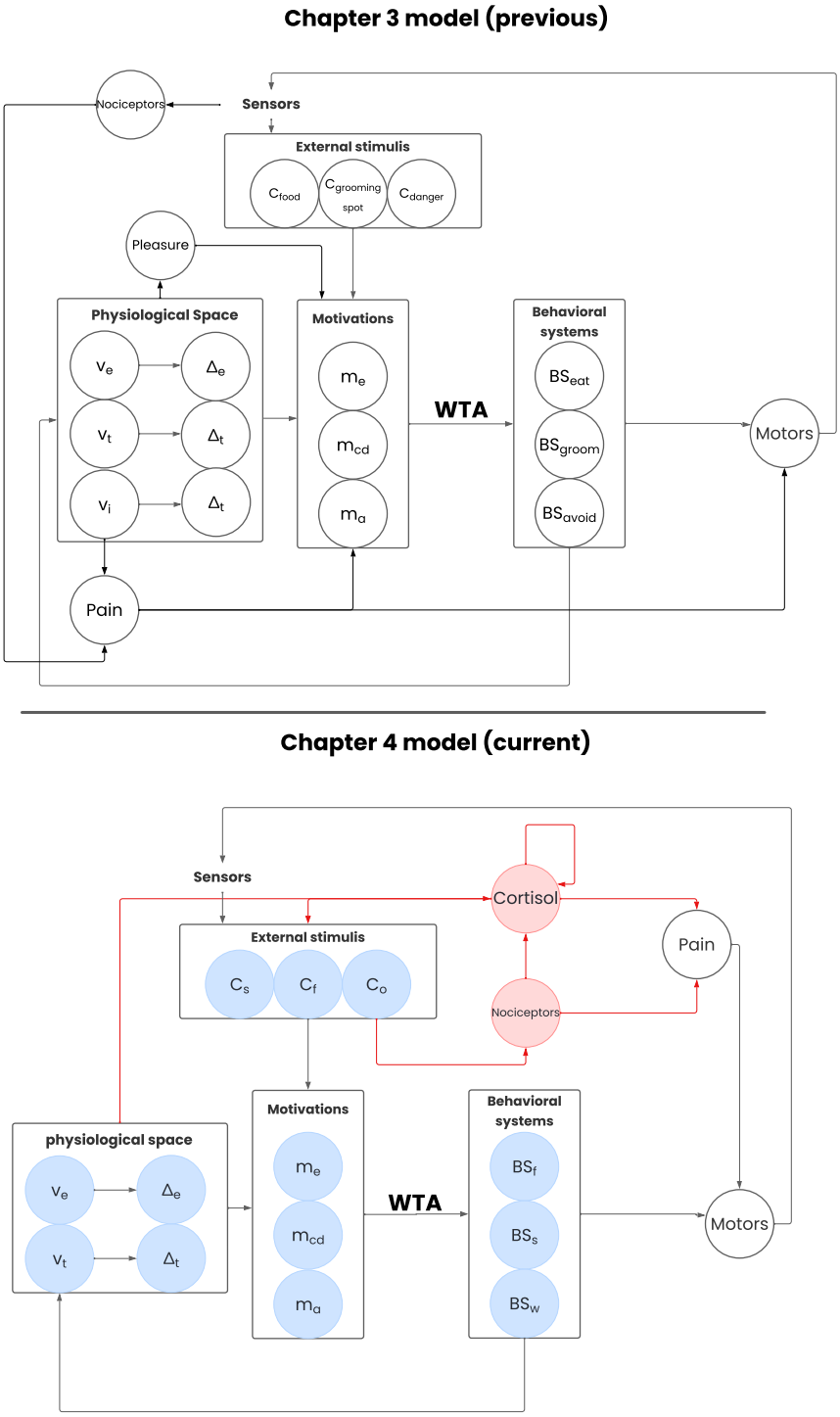


Figure 4.2: Granular view of the motivation-based action selection robot's model compared to the version presented in Chapter 3. Red elements and arrows are the new elements we added and blue elements are the specific elements modified without touching computation and logic Names and elements are specified in Tab. 4.1

Drawing upon the architecture previously described in Chapter 3 I implemented a similar basic action selection architecture using a different robotic platform.

Facing a similar problem where a robot needs to maintain its **physiological variable** within a permitted range, the robot will use **internal and external sensors** that will generate **cues** that will help compute **motivations** which choose a specific action using a **Behavioral system**.

In this specific problem, Khepera will need to maintain energy and temperature within a permitted range. It will use specific elements described in Table 4.1.

Elements	**Name**
Physiological variables (v)	energy, temperature
Sensors (internal & external)	Energy, Temperature, Ground IR Sensors, Nociceptors
External stimuli, or cues (c)	"shade", "food", obstacles
Motivations (m)	Eat, Cool Down, Avoid
Behavioral systems	Consume food, decrease temperature, withdraw

Table 4.1: Elements of the action- election architecture

4.1.2 Physiology

The robot's physiology builds upon the concept of the *physiological space* introduced in Chapter 2, but it extends this concept by allowing each physiological space to have its own ideal value, which may not necessarily be at an extreme. Consequently, the computation of $Phys.error$ differs from the previous chapter.

$$\Delta = \frac{ideal - v}{ideal} \tag{4.1}$$

4.1.3 Behavioral Systems

I proposed to improve behavioral impact on our model by adding main and secondary effects to each behavior. These effects have different impacts on the physiological spaces of the model and are detailed in 4.2.

4.1.4 Sensors (internal and external)

The specific robot we use for this model is a Khepera-IV [97], a K-Team Switzerland made robot desigend for robotic and artificial intelligence research. Khepera-IV is a small, mobile robot designed for research in robotics and artificial intelligence. It has a circular shape and is 5.5 cm in height and 10 cm in diameter.

Behavioral System	**linked motivation**	**Behavior**	**Effects**
Consume food	eat	consume food	↑ energy, ↑ temperature
		seek for food	↓ energy, ↑ temperature
		wander	↓ energy, ↑ temperature
Decrease Temperature	cool down	decrease temperature	↓ energy, ↓ temperature
		seek for shade	↓ energy, ↑ temperature
		wander	↓ energy, ↑ temperature
Withdraw	avoid	withdraw	↓ energy, ↑ temperature

Table 4.2: Behavioral systems, their linked motivations, behaviors, and effects. ↑ indicates that the behavior increase the level of a physiological variable, ↓ that it decrease it level.

I use the following Khepera IV **external sensors** to detect relevant elements of the environment and to create **internal sensors** relevant to specific tasks of the model:

- **Proximity Sensors**: InfraRed (IR) sensors located around the body of the robot are used to detect objects up to 20cm from the robot. For convenience, we'll talk about "Proximity sensors." IR sensors provide values between 1023 (object next to the robot's body) and 0 (object beyond the sensor's range). Proximity sensors are normalized to values in the range 0 to 1.
- **Ambient Light Sensors**: The IR sensors underneath the robot are used to detect the two different types of resources situated on the floor of the arena.
- **Resources sensors**: For our specific action selection problem, the robots need to consume resources to maintain their internal physiological variable within the permitted range. Resources are represented with objects of specific color and texture. Thus, I use ground ambient light sensors to compute **internal sensors**, which will represent the detection of such resources.

4.2 Artificial (simulated) Cortisol

Nociceptors are the sensory nerve cells that respond to potentially noxious stimuli by sending "damage" signals, leading to the perception of pain. Based on previous work on artificial nociception [16], we use the robot chassis as it is "skin" and compute, using infrared proximity sensors, and artificial nociceptors.

Nociceptors are set to detect damage (noxious stimuli) [98, 92]. Sensor values provide us with proximity information that allows us to generate a "proximity field" in which it is possible to reconstruct some information in order to analyze the different noxious stimuli the robot faces. Our artificial nociceptors are sensitive to different types of contact:

- Scratching
- Impact

Artificial cortisol hormone

Cortisol is the primary stress hormone; it has many effects on our body, such as increasing the level of glucose in the bloodstream. It is set to slow the nonessential function in a fight-or-flight [94] situation.

To compute the concentration of cortisol in the "bloodstream," the robot model uses a formula that considers the robot's comfort level and the nociceptor activity. The release rate of cortisol from an "adrenal gland" [99] is computed using the following equation:

$$r_{cortisol} = \alpha \times mean(nociceptors) + \beta \times (1 - comfort) \tag{4.2}$$

Where α and β are constants that determine the relative importance of each input parameter. The $mean(nociceptors)$ term represents the average activity level of nociceptors. To compute the robot's comfort level, we use:

$$comfort = mean(1 - Phys.error) \tag{4.3}$$

Where Δ_i represents the error of each physiological variable, i.e., the difference between its ideal and actual levels (values). The term $mean(1 - phys.error)$ computes the inverse of the error, and taking the mean across all the physiological variables produces a measure of the robot's comfort level.

The effect of cortisol on the robot is twofold. First, it is used to modulate pain perception based on the robot's comfort level and nociceptor activity [100]. The concentration of cortisol is computed using the formula :

$$\begin{aligned} c_{cortisol}(t) = max(1.0, \\ c_{cortisol}(t-1) \cdot \psi_{cortisol} + r_{cortisol} \end{aligned} \tag{4.4}$$

where $\psi_{cortisol}$ is the decay constant.

Additionally, we can also add a **negative feedback** mechanism while computing the gland release rate, having the cortisol concentration influence the new release rate :

$$\begin{aligned} r_{\text{cortisol}} = (\alpha \cdot \text{mean}(nociceptors) \\ + \beta \cdot (1 - \text{comfort})) \cdot (1 + \gamma \cdot c_{\text{cortisol}}(t-1)) \end{aligned} \tag{4.5}$$

Where γ is a constant that determines the importance of the cortisol concentration negative feedback in the release rate.

By prioritizing resource-seeking behaviors that minimize pain and discomfort, the robot can maintain its physiological variables within the permitted range, which is critical for its survival and efficient operation in the environment.

Second, cortisol influences the salience of environmental cues based on the robot's current physiological state and needs. The concentration of cortisol can affect various cognitive and emotional processes, such as attention, memory, and decision-making. By modulating the salience of environmental cues, cortisol impacts the robot's decisions and resource allocation. Our experiments will assess under what circumstances and to what extent this impact can favor (or impair) better decisions.

4.3 Pain perception

A simulated hormone [79] signals the perceived pain. Its perception can be directly correlated to damage or modulated by stress with a specific simulated "cortisol" hormone.

There are multiple interactions between stress and perceived pain; some studies argue that acute stress leads to hyperalgesia [101], and other tends to prove that psychological stress leads to hypersensitivity to pain [102]. This hypersensitivity can be adaptive in stress-related danger environments by strengthening pain perception increase the motivation to act with healing and escaping behaviors [41].

Thus, cortisol, the stress hormone, has an impact on the pain patients perceive. Whether it is still not perfectly clear with different studies which impact it has a twofold impact with hypoalgesia and hyperalgesia that can be modeled similarly as a cubic curvilinear function [103]. Its impact should be looked at as a multifaceted relationship between acute pain, chronic pain, and stress.

We aim to investigate the impact of simulated cortisol on pain perception in robots. Thus, I propose two ways to model the perception of pain :

- Directly correlated to damage
- Modulated by "cortisol" hormone

Changes in the concentration of cortisol modulate the robot's pain perception and can lead to prioritizing resource-seeking behaviors that minimize pain and discomfort [16].

Pain has an impact on the strength of motor action:

$$speed_{(L/R)} = speed_{(L/R)} \times (1 + 0.5 \times pain(t)) \tag{4.6}$$

When the robot perceives pain, it may reduce the strength of its motor actions to minimize further damage or discomfort. This could affect the efficiency of its resource-seeking behaviors, and the robot must balance the need for resource acquisition with the need to avoid pain and damage.

Cortisol and perceived pain influence on the specific action selection robot architectures are summarized in (Fig. 4.2).

4.4 Experiments

We present the results of a series of experiments designed to evaluate the viability and evolution of our robot in an environment with different numbers of predators. We also compare the results of using cortisol to modulate pain perception with those of using only nociception in response to predator attacks.

Our hypothesis is that cortisol modulation of pain perception in robots can enhance their ability to adapt to their environment. Negative feedback on the release rate will also improve the ability to adapt by adding habituation mechanisms that will lead. By modulating the experience of pain associated with predator attacks, robots may be able to more effectively escape or defend against predators, improving their chances of survival.

The primary research question we aim to address in this experiment is: How does stress and cortisol modulation compare to nociception alone in enhancing a robot's survival capabilities in a predator-rich environment? To answer this question, we have designed a protocol to test our robot under different conditions in a Two-Resource Problem (TRP) environment, where it needs to satisfy the need to consume two resources (food and shade) timely to maintain energy and temperature at a certain level for survival.

4.5 Experimental setup

Our robot was tested in the context of a Two-Resource Problem (TRP), where it needs to satisfy the need to consume two resources (*food* and *shade*) in order to maintain *energy* and *temperature* at a certain level to stay alive.

The environment (Fig.4.3) consists of a $1.5m$ by $1.5m$ wooden arena containing two types of resources represented by floor tiles of different textures, which the robot can detect and discriminate between using its IR floor sensors. Resources are symmetrically distributed in this environment.

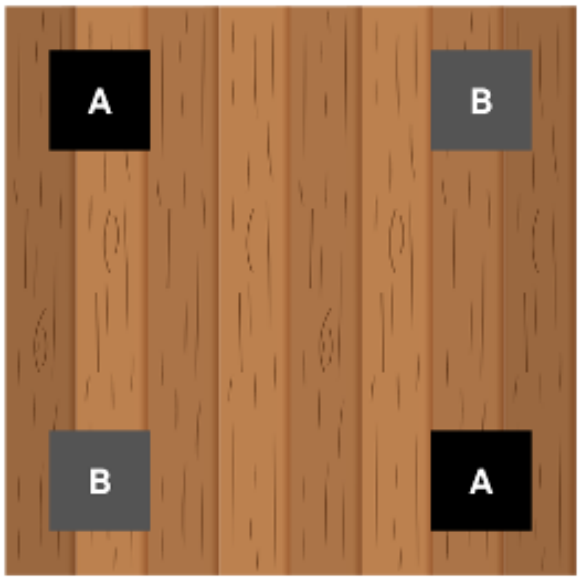


Figure 4.3: 1.5m by 1.5m wooden arena

Number of predators	**Pain perception**	**Type of predators**
0	Damage-correlated	Stalking
1	Cortisol-modulated	Agressive
2	cortisol-modulated with negative feedback	Repettitive Agression
3		

Table 4.3: Different conditions tested

We've embedded our architecture into a Khepera IV robot, characterized by its circular shape, 5.5 cm height, and 10 cm diameter, equipped with a variety of sensors.

To introduce environmental variability and stress-related danger, we incorporated robots exhibiting predatory behaviors. For this purpose, we utilized Thymio-II robots (www.thymio.org). These predators have been programmed with various behaviors, including stalking, aggressive prey-searching, and repetitive contact. Specifically, the "stalking" behavior allows the Thymio-II robots to move slowly, avoid edges, and, upon detecting another object or robot, pursue and attempt to "attack" it. Crucially, upon contact with the Khepera-IV robot, the Thymio-II continues its stalking, presenting a continuous threat. The "aggressive" behavior sees the predator actively searching for and trying to damage the Khepera-IV. Meanwhile, the "repetitive" behavior ensures the predator contacts the Khepera-IV at regular intervals.

These predator implementations provide a controlled and consistent predatory environment. This setup enables us to study the effects of varying predation levels on pain perception, resource-seeking actions, and the overall survival of the Khepera-IV robot.

To investigate how different levels of stress-related danger can impact pain perception and how stress and cortisol can enhance survival capabilities, we will conduct a series of experiments testing different conditions. The different tested conditions are the ones listed in Tab. 4.3.

We will test 5 runs for each experimental condition (90 in total). Each run lasts 600s (or less if robot "dies" (i.e. one of it physiological variable goes outside the permitted viability range)).

In order to evaluate the viability of our robot model and its survival capabilities, we will assess different metrics :

- **Survival rate** after 600s
- **Comfort** (management of internal variables)
- **Cortisol** level
- **Pain perception**

- **Activity Cycles** (errors tracked in physiological space)
- **Motivation** intensities

4.5.1 Live interface

We implemented on Khepera-IV a live interface to display the different informations about sensory inputs, evolution of the internal variables and state of the robot. We also added motor command and control of the model in the live interface to provide a live demo environment Fig. 4.4.

This web interface is implemented using a using a JavaScript library that communicate with python model through websockets.

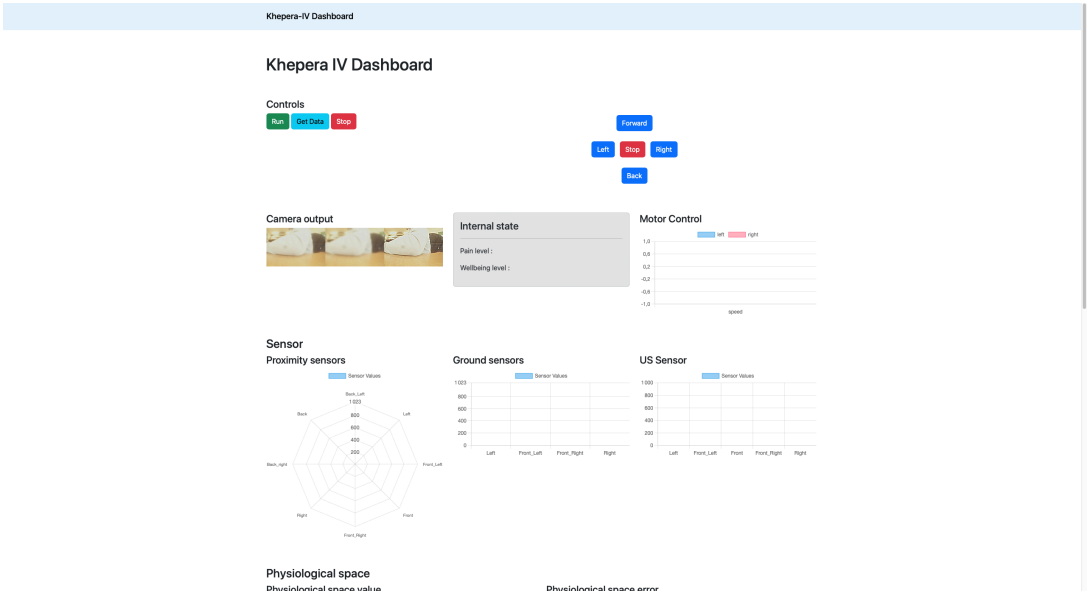


Figure 4.4: Screenshot of the webpage where we can control robot and display informations about the model such as sensory inputs, camera feedback

4.6 Results and discussion

Among the variety of results we could get from our experiments some appeared interesting to observe to evaluate viability of our model.

4.6.1 Viability of the model

First of all, assessing, the viability, or survival rate after 600s, is useful to understand the survival advantages of different pain perceptions.

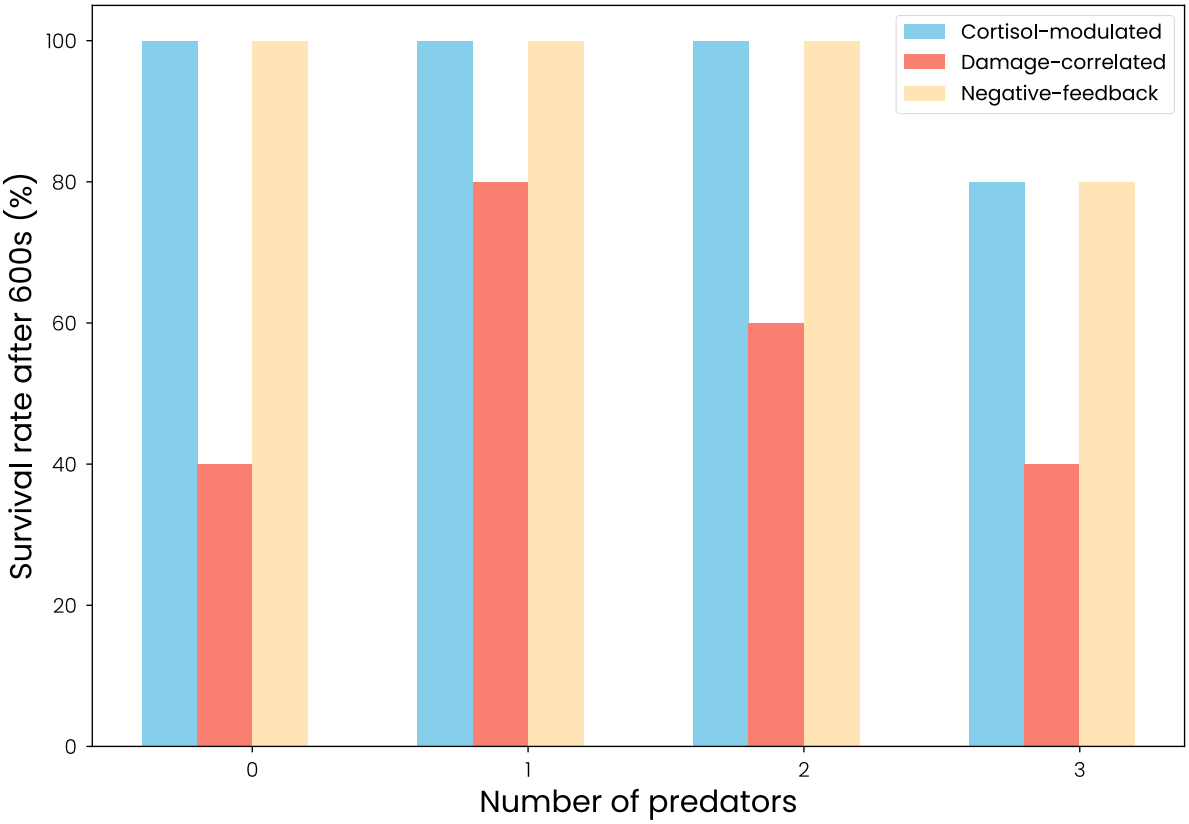


Figure 4.5: Survival rate after 600s, with 0 to 3 stalking predators. Blue represents viability for cortisol-modulated pain perception, orange for damage-correlated pain perception, and yellow for negative feedback pain perception.

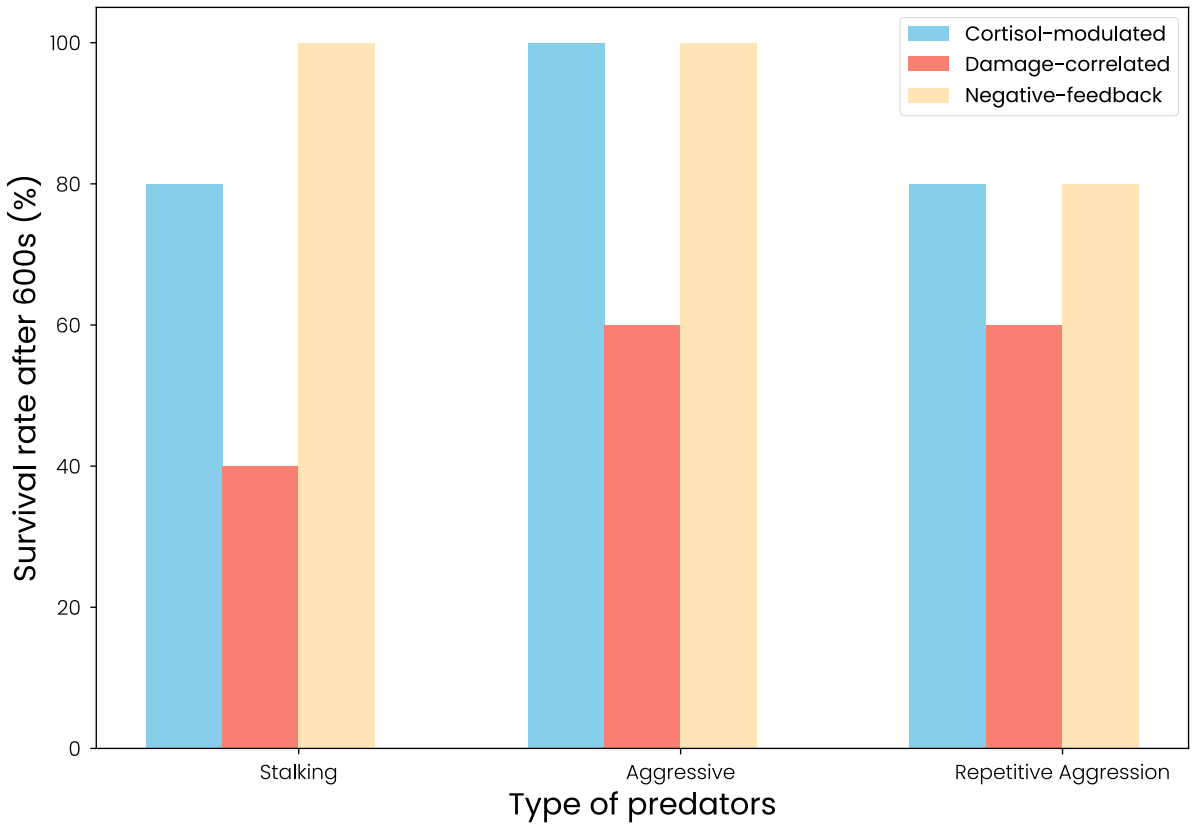


Figure 4.6: Survival rate after 600s with two predators, varying the type of predation (stalking, aggressive, repetitive aggression). Blue represents viability for cortisol-modulated pain perception, orange for damage-correlated pain perception, and yellow for negative feedback pain perception.

Examining Fig. 4.5 and Fig. 4.6, which depict the robot's survival rate over five runs for each condition, several observations can be made. Firstly, in the absence of stress-related predation danger, the method of pain perception computation does not impact viability. For all predator conditions (both numbers, ranging from 1-3 and type), cortisol-modulated pain perception yields higher survival rates than damage-correlated pain perception. Secondly, this advantage in viability is further augmented with negative feedback.

4.6.2 Hormone Secretion Dynamics Graph

Our analysis of the Hormone Secretion Dynamics Graph reveals in "Fig. 4.7" distinct clustering patterns for each scenario (1 to 3 predators), providing valuable insights into the interplay between cortisol concentration and release rate in the context of varying predator presence. To determine the number of clusters for each scenario, we used the elbow method and x-mean. In a scenario with 3 predators, the distribution appears uniform, while with 2 predators, we observe two clusters: one with high cortisol concentration and low release rate, indicative of memory's impact on hormonal concentration, and another with both low concentration and release rate, as well as a region where concentration is low, and release rate, is high. For scenario one, predators, two clusters emerge, with a

smaller one exhibiting high concentration and low release rate and a larger one characterized by low concentration and high release rate. This pattern suggests that cortisol exhibits lower memory dynamics when fewer predators are present, a trend that is further confirmed by the clustering observed in scenarios with no predators, where only the cluster with concentration and high release rate.

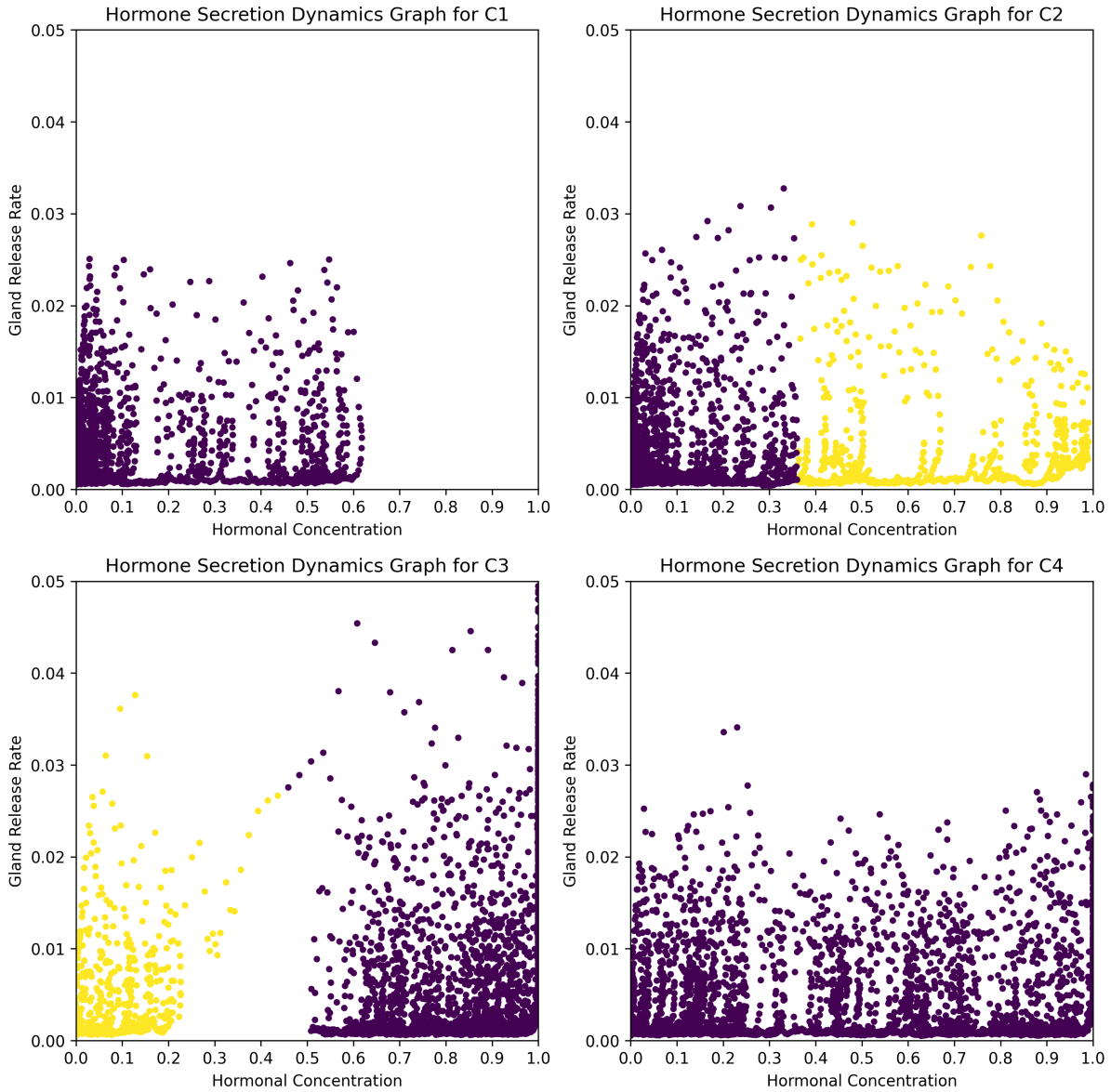


Figure 4.7: Hormone secretion dynamics graphs for scenarios with (A) 0, (B) 1, (C) 2, (D) 3 Stalking predators. Hormonal concentration is compared to gland release rate to understand the hormone evolution in specific scenarios

These findings emphasize the role of cortisol in modulating the robot's perception of pain and stress response, with varying memory dynamics based on the number of predators in the environment. By adapting its hormonal regulation in response to environmental challenges, the robot is able to demonstrate enhanced performance and adaptability.

4.6.3 Activity Cycles in the physiological space

Physiological variables delineate a physiological space, sometimes termed as a "viability zone" [104, 105], wherein survival is ensured. They are straying outside these defined boundaries, resulting in death. In our TRP, this space is two-dimensional, with each axis indicating the deficit of a specific physiological variable. [12] Activity Cycles (AC) track the evolution over time of the deficits within the physiological space, which represents, in the physiological space, the behaviors executed to reduce those errors.

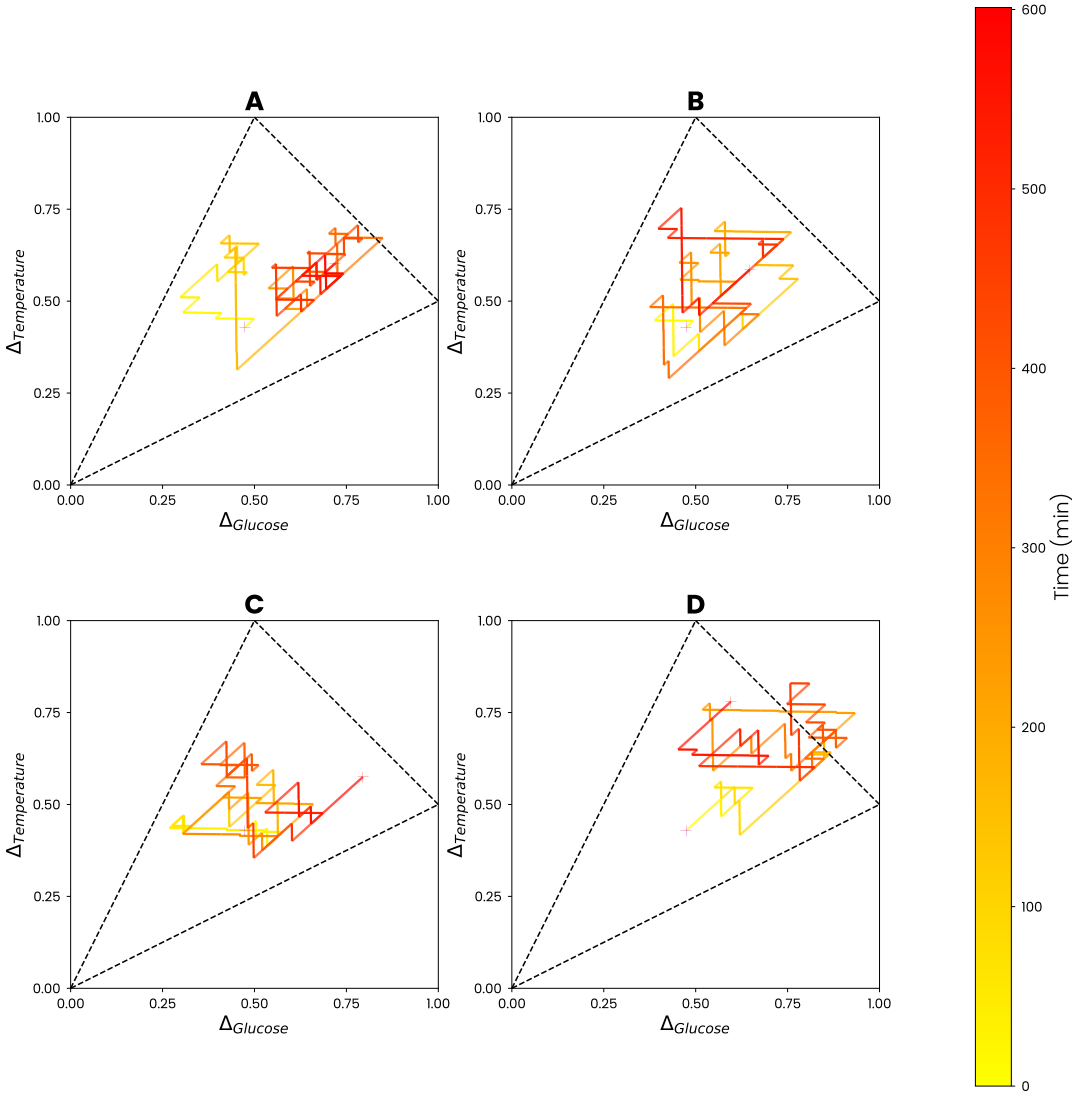


Figure 4.8: Activity cycles for the damage-correlated pain perception with (A) 0 predators; (B) 1 predator; (C) 2 predators; (D) 3 predators, $\Delta_{Temperature}$ is compared to Δ_{Energy} triangle shape represents the viability stability cycles, going outside its area represents danger (with high deficits for one or both variables, respectively) in the physiological space

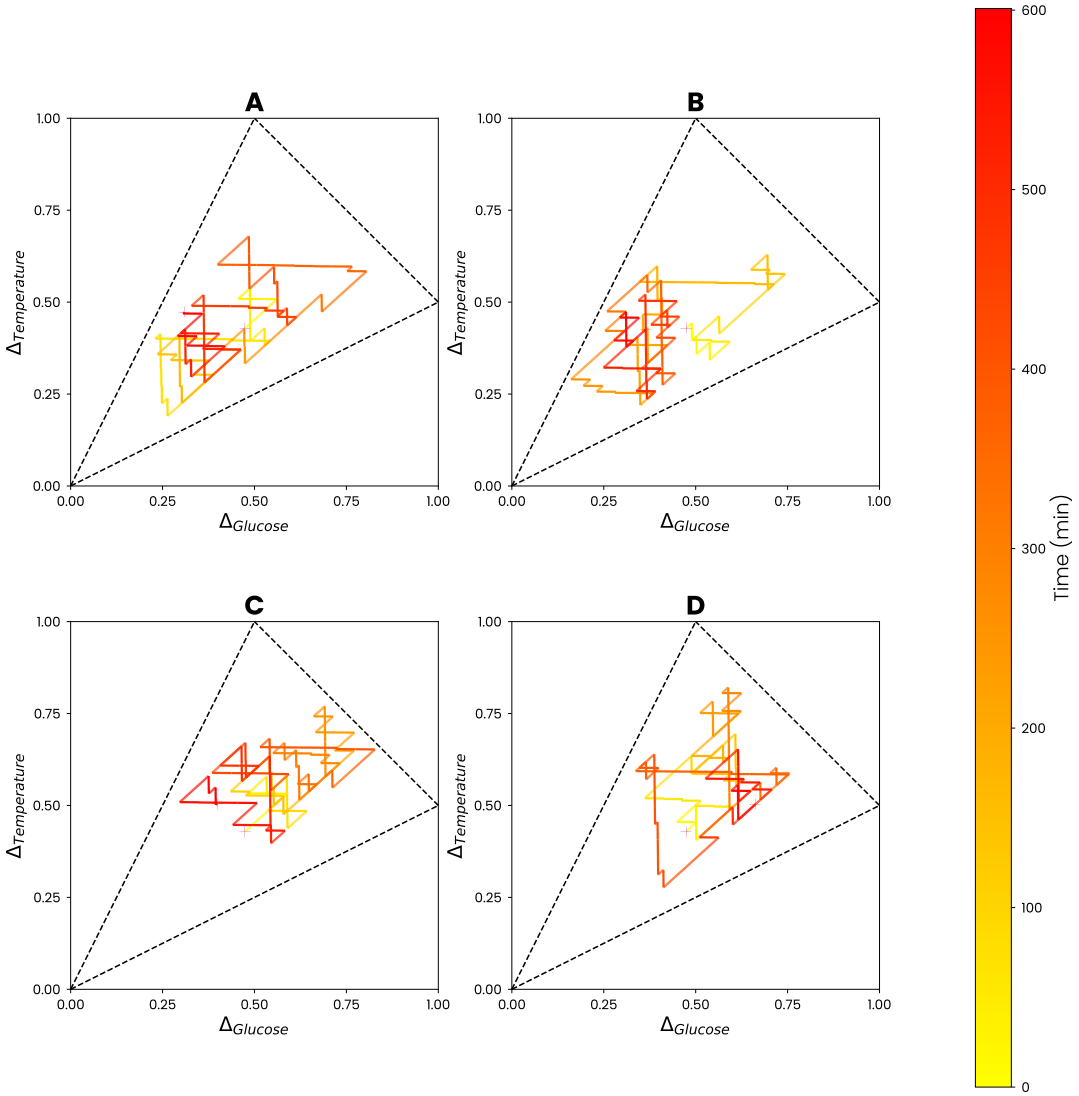


Figure 4.9: Activity cycles for the cortisol-modulated pain perception with (A) 0 predators; (B) 1 predator; (C) 2 predators; (D) 3 predators, $\Delta_{Temperature}$ is compared to Δ_{Energy} triangle shape represents the stability cycles, going outside its area represents danger (with high deficits for one or both variables, respectively) in the physiological space

Examining Fig. 4.8 and Fig. 4.9, we note that in environments with a high level of stress-related danger (represented by 3 predators), damage-correlated pain perception results in less balanced deficit management. Errors not only surpass the viability safe zone but also venture into dangerous regions within the physiological space. Additionally, a review of Fig. 4.10 reveals that the activity cycles tend toward more sustainable management of physiological deficits.

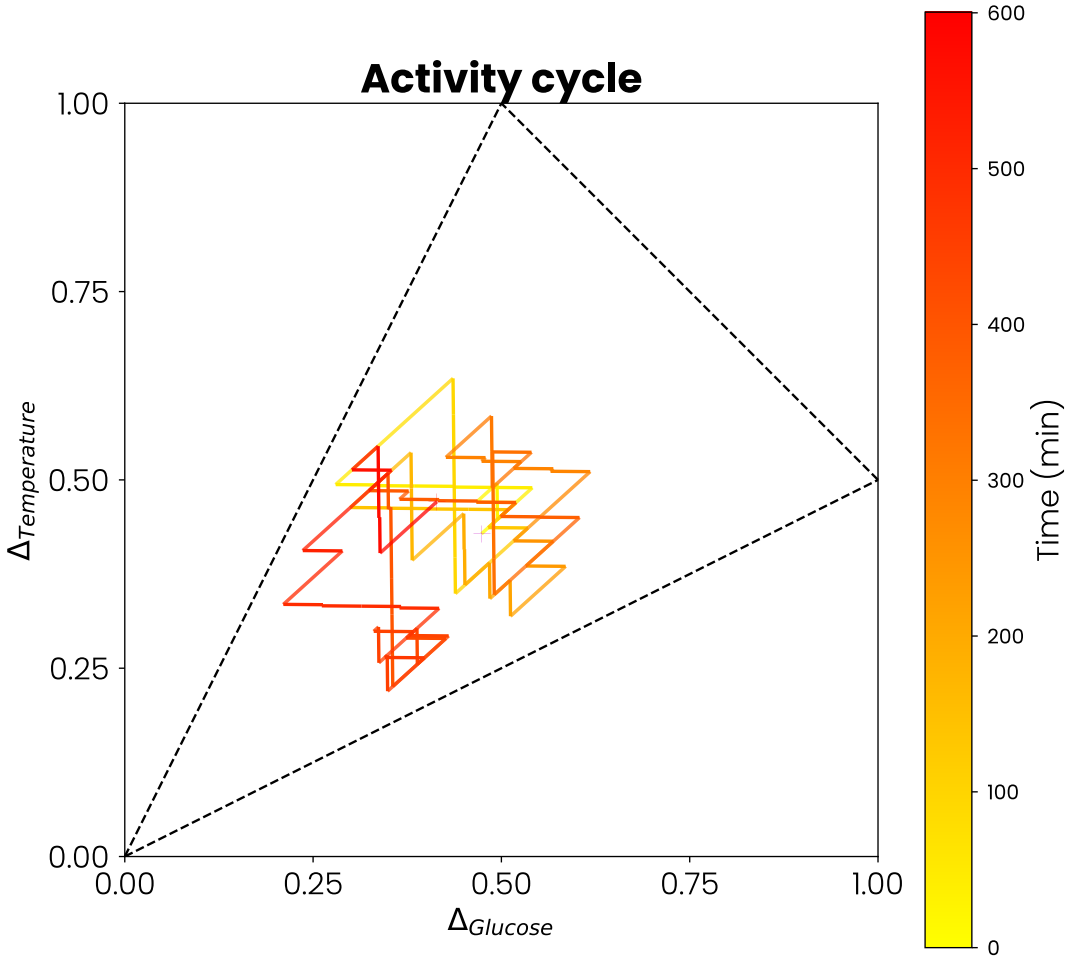


Figure 4.10: Activity cycles for the cortisol-modulated with negative feedback pain perception with two predators, $\Delta_{Temperature}$ is compared to Δ_{Energy} triangle shape represents the stability cycles, going outside its area represents danger (with high deficits for one or both variables, respectively) in the physiological space

4.6.4 Intensity of cortisol over time

In various scenarios, we analyzed the cortisol intensity over time. As depicted in Fig. 4.11, in low-stress environments (e.g., without predators), cortisol is released intermittently. However, in environments with increased stressors, such as two predators, cortisol levels not only elevate but remain comparatively high, punctuated by occasional dips.

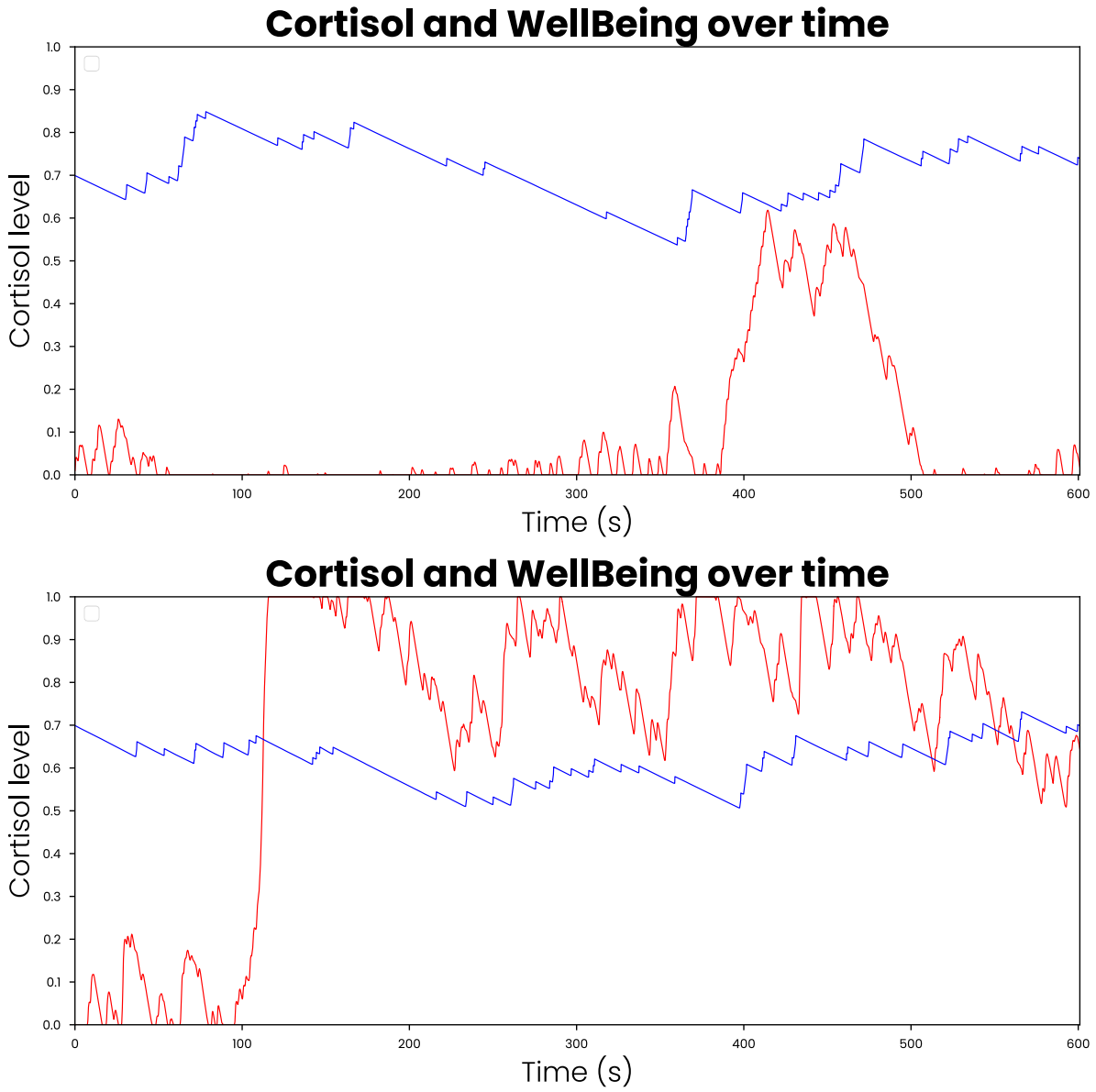


Figure 4.11: Intensity of Cortisol and Wellbeing (the inverse of the mean of the error of the physiological variables) over time with cortisol-modulated pain perception in scenarios with (top) 0 and (bottom) 2 predators.

Moreover, examining Fig.4.12, we see the emergence of **sensitization** and **habituation** mechanisms due to the negative feedback in cortisol-modulated pain perception. Specifically, in low-predation stress environments, if no early-life stress is experienced, habituation is evident, and cortisol levels remain substantially lower compared to situations with the exact predator count but cortisol-modulated pain perception. Conversely, in high-predation stress environments, early-life stress results in sensitization, leading to sustained high cortisol levels throughout the robot's "life."

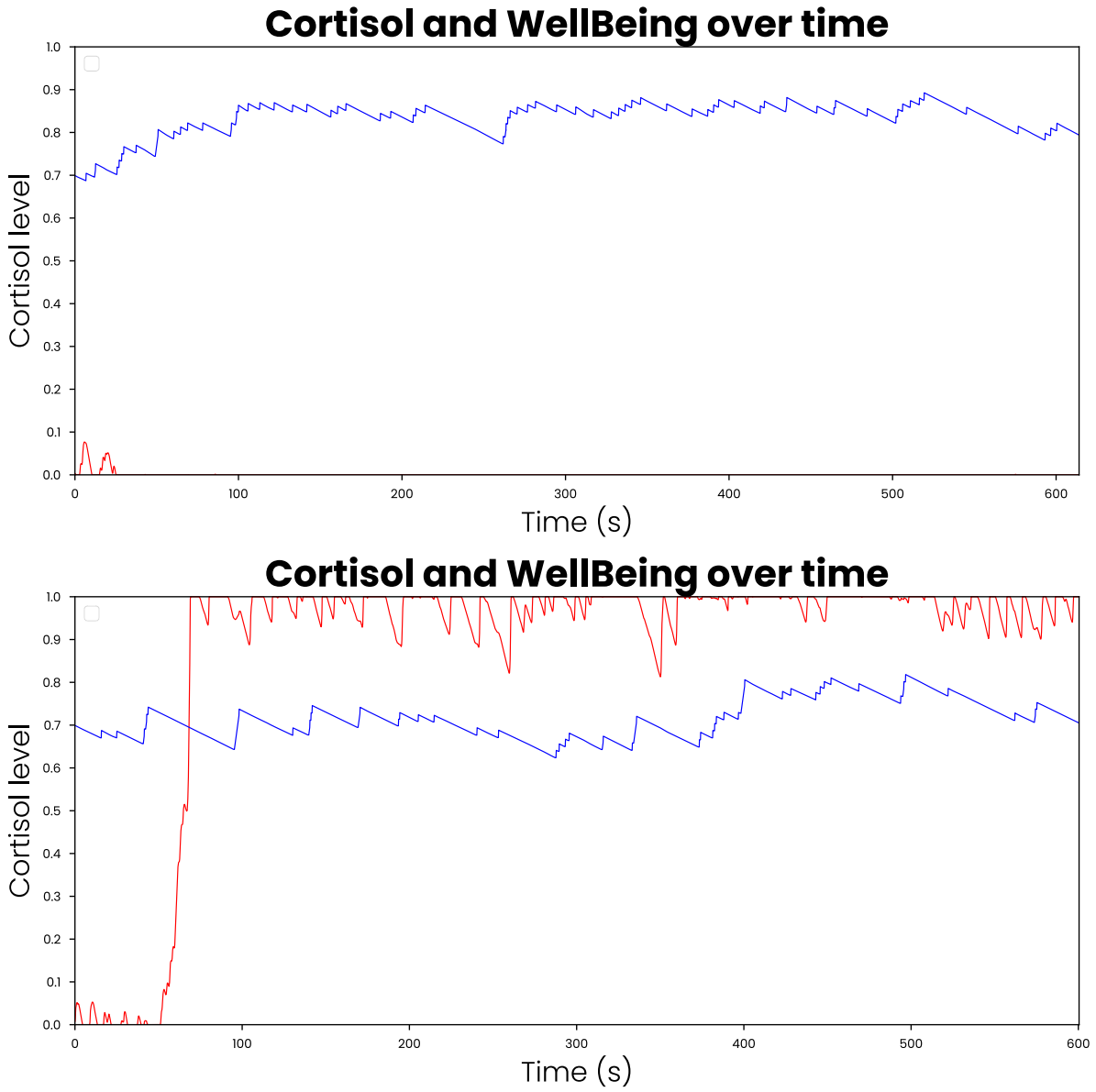


Figure 4.12: Intensity of Cortisol and Wellbeing (the inverse of the mean of the error of the physiological variables) over time with cortisol-modulated and negative-feedback pain perception in scenarios with (top) 0 predators and (bottom) 2 predators.

4.6.5 Intensity of motivations over time and nociception: the example of cortisol-modulated pain perception with two predators

In our experiments, we explored the interplay between nociception and the selection of motivations in our cortisol-augmented robot model. Taking scenario C3 as an example, where the robot faces two predators in the arena, we observed in Fig. 4.13 and Fig. 4.14 a strong correlation between nociceptor activation and the choice of motivations. As depicted in the "Intensity of Motivations Over Time" graph and the nociception activity

graph, we can see a significant increase in nociceptor excitation around the 110-second mark. During this phase, the robot consistently selects the danger motivation for an extended period.

The elevated nociceptor activity corresponds to the emergence of an attack behavior, wherein the robot confronts and engages with predators while simultaneously activating some of its nociceptors. Interestingly, when multiple nociceptors are excited, the robot prioritizes the danger motivation over other motivations, such as hunger or cold. This finding highlights the adaptability of our robot model to varying environmental conditions and threats.

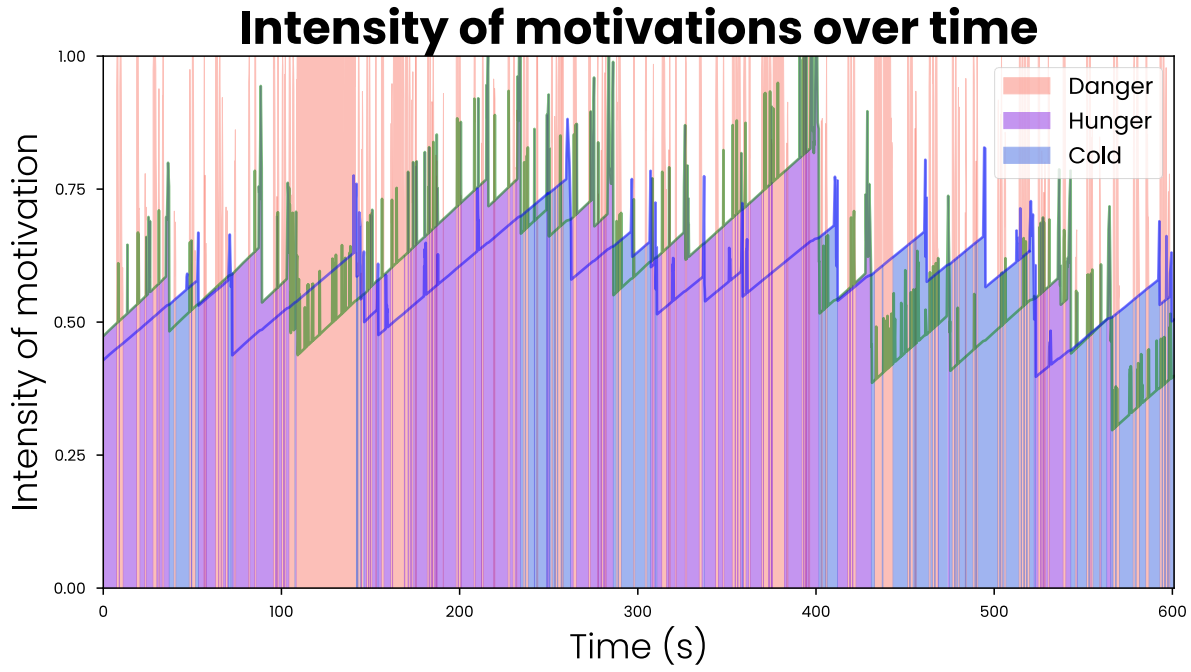


Figure 4.13: Intensity of motivations over time for cortisol-modulated pain perception with two predators.

4.6.6 Emerging behaviors

In our cortisol-modulated robot model, two distinct behaviors emerged.

First, in scenarios with cortisol-modulated pain perception with a high level of stress-related predation, where the number of stalking predators is higher, the robot appears to engage in a "fight" response, characterized by executing left and right turning loops and occasionally making contact with the predators, repelling them. This behavior is more prominent when cortisol levels are elevated. The integration of cortisol in the action selection architecture serves to modulate the robot's perception of pain, analogous to the stress response in biological organisms. As cortisol levels increase, the robot's aversion to pain diminishes, allowing it to engage in riskier behaviors.

In Fig. 4.15, we can observe an example of this behavior during scenario C4. In this particular moment, the robot is chased by two predators, and the third one is coming in

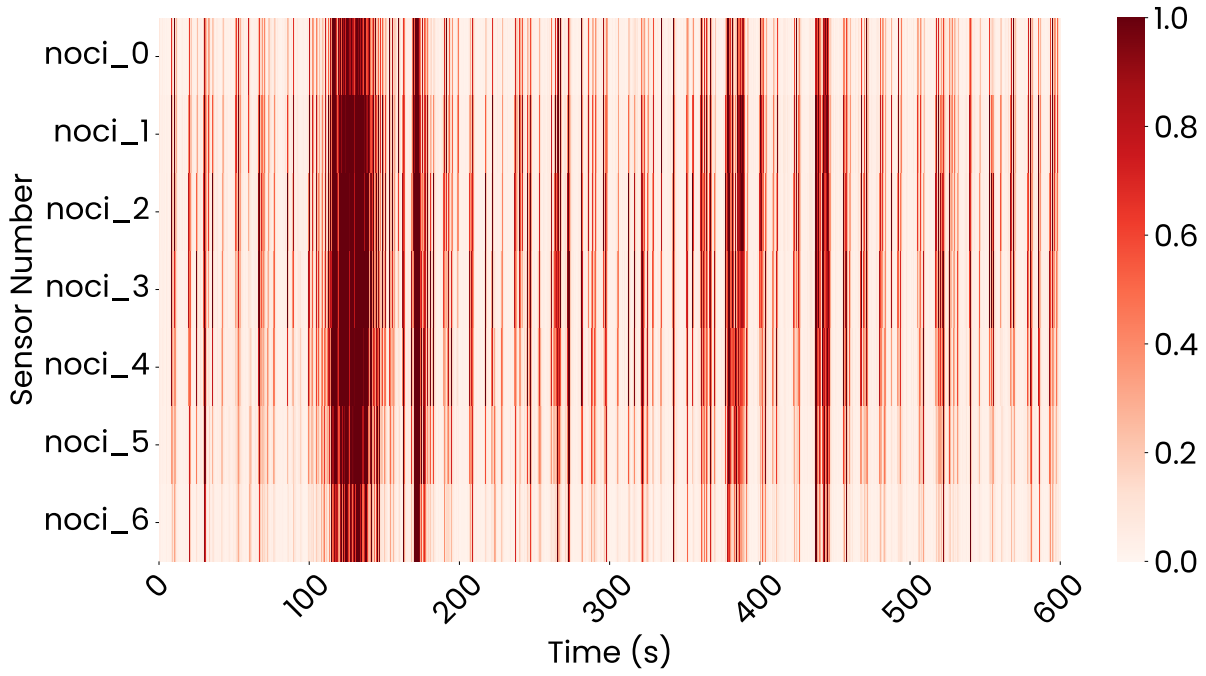


Figure 4.14: Nociceptors Heatmaps for cortisol-modulated pain perception with two predators.

its direction. The Khepera robot is blocked and will make contact that will repulse one of the three predators, allowing it to escape the first attack of the two predators.

Figure 4.15: Emerging attack behavior in cortisol-modulated pain perception with three predators : (A) the robot is confronted by two predators blocking a resource. (B) the robots turn in the direction of the predators. (C) The robot repulses one of the predators, accessing the resource. (D) The robot turns to escape.

Figure 4.16: Emerging laziness behavior in cortisol-modulated with negative feedback with one predator: (A) the robot is turning on itself stuck to the resource, a predator is approaching, (B) predator is getting closer to the robot, still turning on itself close to the resource, (C) predator is attacking robot, robot stop to turn on itself, (D) robot is escaping from danger staying close to resource

This may be interpreted as an adaptive strategy to cope with the high-threat environment, wherein the robot prioritizes self-preservation and resource acquisition by actively confronting predators rather than solely avoiding them.

Second, another pattern seen with cortisol-modulated with negative feedback pain perception is a "laziness" behavior (Fig. 4.16). In specific scenarios, robots linger near a resource, circling it until a predator disrupts this repetitive pattern. This happens when the robot effectively manages its physiological resources. With low cortisol and physiological deficit levels, the robot's motivations to address physiological needs decrease, resulting in the predominant withdrawing motivation that triggers this "circular" behavior. Such behaviors may reflect aspects of human and animal physiology; when basic needs are met, there may be no pressing desire to seek out or consume more resources.

4.7 Discussion

The exploration of cortisol-modulated pain perception in robotic systems, particularly within the context of varied predating environments, has produced some results we can discuss. Our study on the impact of simulated cortisol in a two-resource decision-making scenario highlights the hormone's critical role in enhancing robotic adaptability to evolve and select correct action, which is crucial in dynamic environments with varying predator types and numbers.

First, the model's viability improvement shows the simulated hormone's influence on the model. Indeed, the impact of hormones on decision-making systems, and particularly hormonal feedback, has led to sensitization and habituation. The hormone acts as a memory for the system, allowing it to evolve differently depending on the contexts. This mechanism is beneficial if coupled with a learning logic.

The study also made important observations about the emergence of emergent behaviors. Whether predators cornered the robot and began to fend off attacks to flee or, in a safe environment, began to linger around a resource, we observed that extreme cases led

to diametrically opposed behaviors, highlighting the interest in the simulated hormonal mechanism.

Finally, incorporating cortisol-modulated pain perception into robots improved their survival rates and led to more efficient management of physiological variables, underlining the potential of biologically inspired mechanisms in improving model responses to environmental contingencies.

Looking ahead, our research opens several avenues for future work. A key area of interest is investigating long-term sensitization effects and early-life experience on pain perception in robots akin to chronic pain in humans. This includes studying the impact of varied temporal dynamics and experimenting with alternative computational methods to understand further and enhance the robustness and flexibility of pain perception in robotic systems. Such research could deepen our understanding of pain and perception and simulate the cortisol hormone impact in artificial agents such as robots.

Chapter 5

The development of embodied sensory processing and cortical representations through tactile interaction

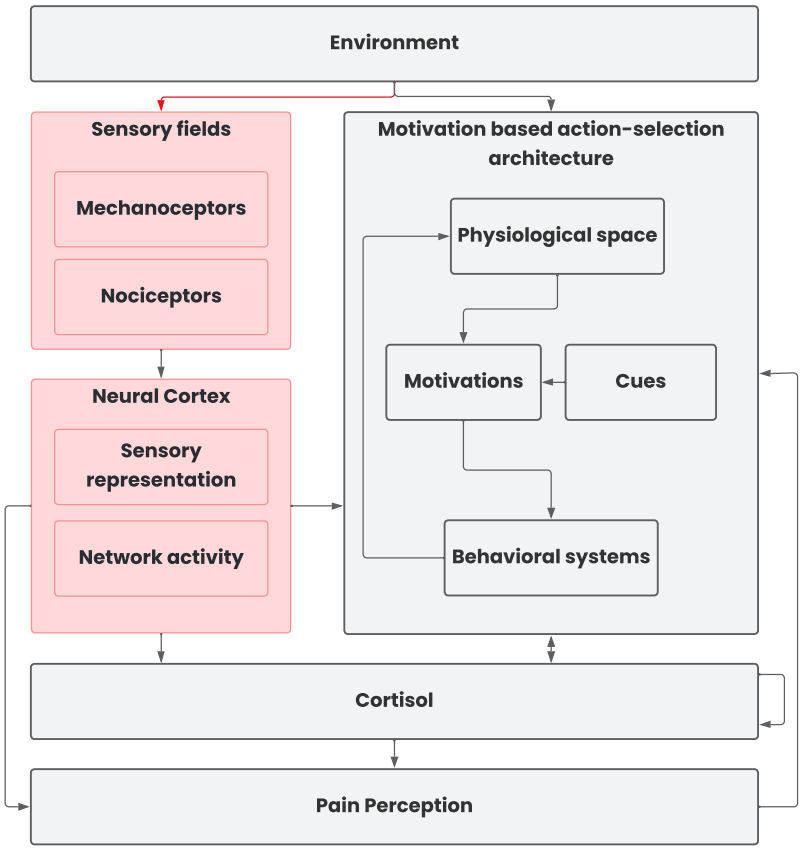


Figure 5.1: High-level diagram of our model and the effects we investigate. Red areas and arrows indicates the elements developed in this chapter.

Touch is a sensory modality that allows organisms to perceive and respond to physical contact with their environment through specialized receptors in skin and other tissues. It

is essential for constructing internal representations of the world, enabling the detection of noxious stimuli that signal harm. It also supports spatialization of the body helping to understand its position and movement in space. Touch perception involves sensory inputs such as mechanoception and nociception.

This predefined pathway from sensory receptors to precise areas of the cortex could be at the origin of the brain's spatial and functional modular organization, where neurons and regions associated with common modalities or functions are more strongly connected [106]. More precisely, plasticity seems to shape these neural assemblies associated with specific sensory modalities or features within a modality, under the action of co-activation zones [107].

Building on my studies on pain modeling in robots, and on Raphaël Bergoin's thesis on inhibitory plasticity in neural memory formation [108], the initial motivation for this study was to understand and model the emergence of a complex sensory representation of touch combining a new representation of mechanoception and nociception in a "sensory body" of a mobile robot, and a neural network model of the anterior cingulate cortex (ACC) and somatosensory cortex in an immature brain (i.e. not having reached its definitive organization). More precisely, the aim is to model the pathway from nociceptors (respectively mechanoreceptors) associated with the skin, to the ACC (respectively somatosensory cortex).

To this end, we use a Khepera IV robot (http://www.k-team.com/khepera-iv). The robot chassis can be considered as a "human skin", a metaphor that overlooks the fact that the input that robots can process, often limited to proximity sensors like IR or ultrasonic, as in our case, is very far from the complexity and the nuanced information human skin provides, such as texture, temperature, and pressure variations. Although efforts have been made to develop sensors mimicking biological features [63, 62], they may not align with the current capabilities of robots [109]. Although we use the IR sensors fitted around the robot's chassis to map the receptive field of the robot's body, we have developed a complex representation of touch (mechanoception and nociception) in the "sensory body" of the robot, in order to extract relevant tactile features to be transmitted to a biologically realistic artificial neural network. This neural network will then adapt to these external signals, shaping its overall organization.

5.1 The sensory body

This section describes the robot's "sensory body" used to model tactile sensations and the interaction between mechanoreceptors and nociceptors.

5.1.1 Tactile sensory fields

To model complex tactile sensing in our robot, we have developed a "sensory field" using proximity sensors, to better capture some of the complexity of human skin's tactile sensations. It is conceptualized through both its nominal and actual forms, where sensor readings, relative to a nominal (undisturbed) position, assess deformations triggered by

environmental interactions. This analysis not only quantifies these interactions but also provides a qualitative insight into the robot's tactile experience.

We employ the Khepera-IV robot, a compact circular robot fitted with eight evenly distributed InfraRed (IR) sensors around its chassis. To allow for a more detailed spatial analysis, we first interpolate additional values between the IR—the average of the readings of two consecutive sensors is interpolated twice between each pair of consecutive IR sensors, to enhance spatial resolution from 8 to 32 values. These interpolated values are then translated into polar coordinates, offering a nuanced understanding of sensory interactions.

$$\{(r_i, \theta_i) | i \in \{1, 2, \ldots, n\}\} \tag{5.1}$$

$$r_i = (1 - v_i) \cdot \alpha + r \tag{5.2}$$

$$\theta_i = \frac{2\pi i}{n} \tag{5.3}$$

With v the sensor value, α the range of the proximity sensor, and r the radius of the robot size. And n the number of sensory values.

For visualization, these polar coordinates are further transformed into Cartesian coordinates, depicting the sensory field as a "blob".

$$\{(r \cdot \cos\theta, r \cdot \sin\theta) \mid (r, \theta) \in \text{polar\_coords}\} \tag{5.4}$$

This representation effectively illustrates the sensory body's "deformation" (its changing, adaptable shape) in response to external stimuli, with a nominal position established to denote its "undisturbed" state (lack of sensory stimulation).

$$d = [\mid d_0 - (1 - d) * d_0 \mid d \in sensor] \tag{5.5}$$

Our model also differentiates between nociceptors and mechanoreceptors, and aims to simulate the skin's elasticity and responsiveness by employing a dual-field approach to encapsulate their distinct responses to stimuli [110]. This distinction is crucial, as mechanoreceptors and nociceptors transmit signals at different speeds: whereas β fibers relay touch and vibration quickly (30-70 m/s), δ fibers, associated with pain, which conduct at slower speeds (5-30 m/s) [51].

5.1.2 Mechanoceptors

Mechanoreceptors detects and interprets a wide range of tactile stimuli encountered by the body [111][112]. They are predominantly located within the dermis layer of the skin (Fig. 5.2 (A)) [4]. If we look at the sensory body we have defined, we can see this elastic skin where the mechanoceptors located in the dermis as the position of the IR sensor values.

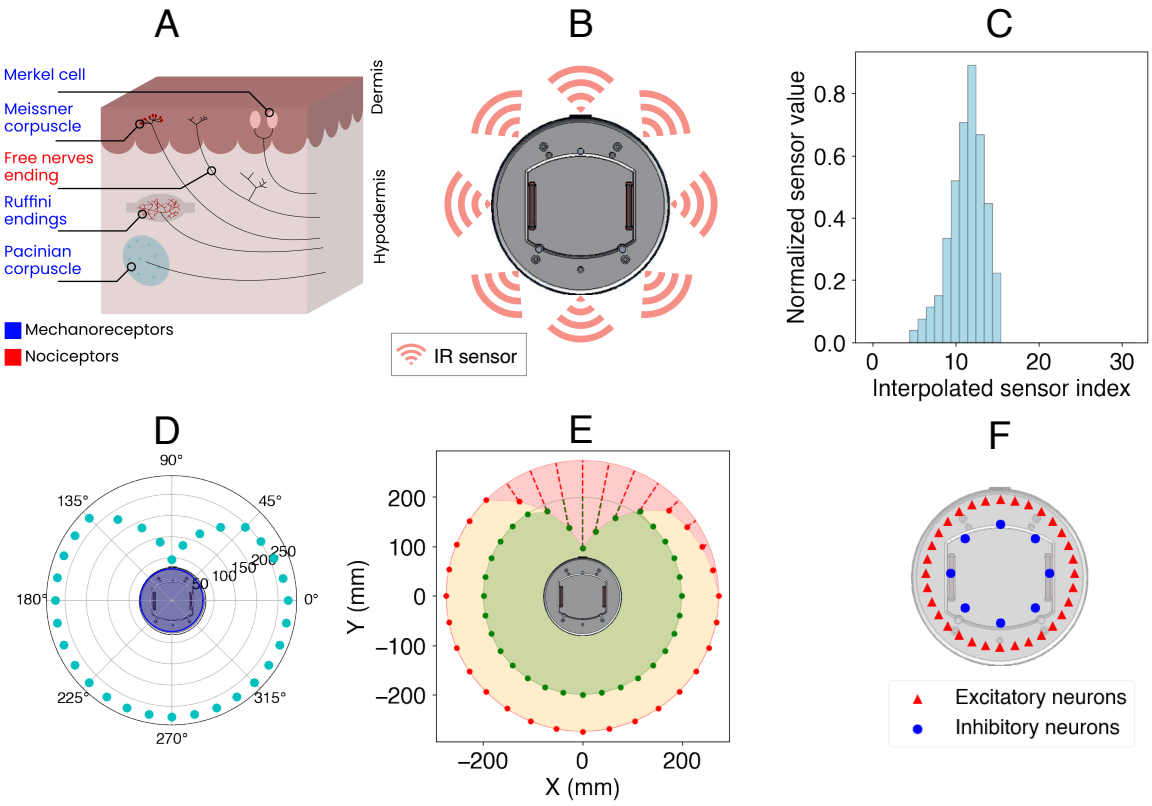


Figure 5.2: (A) Shows four types of mechanoceptors (in blue) and nociceptive free nerve endings (in red) in the human hand, drawn after [4]. (B) Top view representation of a Khepera-IV robot with 8 IR sensors. (C) Normalized and interpolated IR sensor data from 8 to 32 values. (D) Polar coordinates from the data showing the robot's physical outline. (E) Representation of sensory body's deformed through pressure in Cartesian space; the red circle indicates the nominal position of mechanoceptors, the green circle marks the nominal nociceptors' position, illustrating proximity to the robot's body and a positional threshold. The yellow and green fields represent the "deformed" sensory and nociceptive layers, respectively. (F) Cortical neurons processing mechanoceptive information, with red circles for excitatory and blue for inhibitory neurons.

Force The applied force on an elastic material (F) can be calculated using Hooke's Law, $F = k \cdot \Delta L$ where $F \in \mathbb{R}$ is in newtons (N), $k \in \mathbb{R}$ is the material's stiffness constant (N/mm), and $\Delta L \in \mathbb{R}$ is the deformation in mm:

$$d = [\mid d_0 - (1 - d)d_0 \mid] \tag{5.6}$$

Strain Strain (ϵ) is the ratio of the change in length to the original length (L_0):

$$\epsilon = \frac{\Delta L}{L_0} \tag{5.7}$$

Stress Stress (σ) is the force applied to the cross-sectional area of the material:

$$\sigma = \frac{F}{A} \tag{5.8}$$

Young's modulus Young's Modulus (E) is the ratio of stress to strain:

$$E = \frac{\sigma}{\epsilon} \tag{5.9}$$

where E is in pascals (Pa), σ is in newtons per square milimeter (N/mm^2)

The mechanotransduction pathway activates with these receptors' response to external forces, using Young's Modulus at each sensory point to determine activation levels. This data integrates with a neural network simulating the somatosensory cortex's response to tactile stimuli (Fig. 5.2 F). This model encapsulates the complex relationship between physical deformation and neural response, showcasing the encoding and processing of tactile information.

5.1.3 Nociceptors

Nociceptors, recognized as the sensors of pain [85], are sensitive to noxious stimuli, playing a critical role in the body's ability to detect and respond to potentially harmful conditions. These receptors are adept at discerning various characteristics of stimuli, including intensity, duration and even the type of the pain: whether it is sharp, throbbing, or burning.

Based on this understanding, we compute physical information on our sensory field deformation to distinguish touch events, drawing parallels between the nociceptors' functionality and our system's ability to interpret tactile data. This involves analyzing the diffuse or intense deformation of the blob, using a classification algorithm, the velocity of

the touch, its duration, and the frequency of touch encounters. From our sensory body, we thus compute eight pieces of information across four physical aspects (deformation, velocity, frequency and duration), each with two classes, forming an 8-dimensional vector representing noxious stimuli for the neural network. This vector accounts for the speed difference between slower δ fibers (nociceptive) and faster β fibers (mechanoceptive). To account of this speed difference, we temporally delay the sending stimuli to the cortex.

As depicted in Fig. 5.3, our methodology for computing nociceptive features begins by assessing the force exerted across the sensory field to evaluate deformation.

This analysis helps us identify whether the contact is focused or disperse, categorized as intense or diffuse deformation respectively (Fig. 5.2, A & B). In order to do so we will observe the deformation of our sensory field, find and classify the different peaks of deformation that are applied to and the classify the different type of touch.

Algorithm 2 Find and Classify Peaks in Data

Input: Dataset $data$, height threshold H_{thresh}, width threshold W_{thresh}
Output: Lists of intense peaks and diffuse peaks
Initialize $intense\_peaks = []$, $diffuse\_peaks = []$
for $i = 0$ to $len(data) - 1$ **do**
    Calculate previous index: $prev\_index = (i - 1) \mod len(data)$
    Calculate next index: $next\_index = (i + 1) \mod len(data)$
    if $|data[i]| > |data[prev\_index]|$ **and** $|data[i]| > |data[next\_index]|$ **and** $|data[i]| > H_{\text{thresh}}$ **then**
        Initialize $width = 1$
        $left = i$, $right = i$
        while $|data[left]| > |data[prev\_index]|$ **and** $|data[left]| > H_{\text{thresh}}$ **do**
            $width \leftarrow width + 1$
            $left \leftarrow (left - 1) \mod len(data)$
            $prev\_index \leftarrow (prev\_index - 1) \mod len(data)$
            if $left == i$ **then break**
            end if
        end while
        while $|data[right]| > |data[next\_index]|$ **and** $|data[right]| > H_{\text{thresh}}$ **do**
            $width \leftarrow width + 1$
            $right \leftarrow (right + 1) \mod len(data)$
            $next\_index \leftarrow (next\_index + 1) \mod len(data)$
            if $right == i$ **then break**
            end if
        end while
        if $width \leq W_{\text{thresh}}$ **then**
            Append $[width, data[i]]$ to $intense\_peaks$
        else
            Append $[width, data[i]]$ to $diffuse\_peaks$
        end if
    end if
end for
return $intense\_peaks, diffuse\_peaks$

This algorithm allows us to identify the different peaks of deformation applied to our sensory field with the following logic:

- The algorithm iterates over each data point and checks whether it qualifies as a peak by comparing it to its neighboring points (considering a circular array).
- If the point exceeds both neighboring points and surpasses the height threshold H_{thresh}, it is considered a potential peak.
- The width of the peak is determined by expanding left and right from the peak, counting the number of consecutive points that also exceed the height threshold.
- After computing the width, the peak is classified as an *intense peak* if its width is less than or equal to the width threshold W_{thresh}, or as a *diffuse peak* if the width exceeds the threshold.
- The algorithm returns two lists: one containing the *intense peaks* and the other containing the *diffuse peaks*.

After we get these list of vectors we compute the total deformation for both categories, normalizes them, and finally classifies the deformation as either intense or diffuse, depending on which is stronger. If no significant deformation is found, both values are set to zero.

Algorithm 3 Classify Intense and Diffuse Deformation

Input: $blurred\_distance$
Output: $intense\_deformation$, $diffuse\_deformation$
($intense\_peaks, diffuse\_peaks$) ← `find_peaks_and_classify`($blurred\_distance$)
Compute the total deformation for both $intense\_peaks$ and $diffuse\_peaks$
Normalize deformations based on peak count
if any deformation is detected **then**
    Compare $intense\_deformation$ and $diffuse\_deformation$
    Set the dominant deformation to 1 and the other to 0
else
    Set both deformations to 0
end if
return $intense\_deformation$, $diffuse\_deformation$

We then determine touch activation based on these deformations (Fig. 5.2, C) :

$$E(t) = \begin{cases} 1 & \text{if } S(t) > treshold \\ 0 & \text{otherwise} \end{cases} \tag{5.10}$$

Where $E(t)$ is a touch event. With this events we compute key tactile characteristics such as frequency of touch events (Fig. 5.2, D & E).

$$N = \sum_{i=1}^{n} \mathbb{I}_{E_i}(t) \tag{5.11}$$

$$f = \frac{N}{dT} \tag{5.12}$$

Where $\mathbb{I}_{E_i}(t)$ is the indicator function of touch events (i.e. a function that takes the value of 1 if the event E_i occurs at time t, 0 else).

We also compute duration (Fig. 5.2, I & J).

$$D_i = t_{end} - t_{start} \tag{5.13}$$

We also compute the velocity of deformation from the sensory field signal. Once we get the velocity we classify and apply a nonlinear function, based on a hyperbolic tangent, to compute the value of the classified signal. this allows us to ensure a gradual transition of velocity as the signal increases or decreases behind the selected threshold for velocity classification (Fig. 5.2, G & H).

$$v(t) = \frac{dS(t)}{dt} \tag{5.14}$$

5.2 Neural network model

In this section we summarize the model built by Raphaël Bergoin [108]. To process sensory information from mechanoreceptors and nociceptors and to model the neuronal activity of the somatosensory cortex and the ACC, we employ a biologically inspired spiking neuronal network subject to synaptic plasticity. For more details on the model and implementation choices, see [108, 113].

5.2.1 Spiking neuronal network model

Throughout this study, we use a network of excitatory-inhibitory heterogeneous quadratic integrate and fire (QIF) neurons [114]. The network is composed of 80% of excitatory neurons and 20% of inhibitory neurons, as commonly accepted in the human cortex [115]. The inhibitory neurons are divided into two distinct populations: a population following Hebbian learning, and a population following anti-Hebbian learning (i.e. neurons that fire together, decoupled together).

Therefore, the evolution of the membrane potential V_i of each neuron ($i = 1, ..., N$) is described by the following equation:

$$\tau_m \dot{V}_i = V_i^2(t) + \eta_i + g_e S_i^e(t) + g_{hi} S_i^{hi}(t) + g_{ai} S_i^{ai}(t) + I_i(t) + \xi_i(t), \tag{5.15}$$

where synaptic inputs $S_i^e(t)$, $S_i^{hi}(t)$, and $S_i^{ai}(t)$ (excitatory, Hebbian inhibitory, and anti-Hebbian inhibitory, respectively) for neuron i are defined by:

$$\tau_d^{e(i)} \dot{S_i^{e(hi,ai)}} = -S_i^{e(hi,ai)} + \frac{\tau_d^{e(i)}}{N_{e(hi,ai)}} \sum_j^{N_{e(hi,ai)}} w_{ij}\delta(t - t_j), \tag{5.16}$$

where $\tau_m = 0.02$s is the membrane time constant, $\tau_d^e = 0.002$s and $\tau_d^i = 0.005$s the time decay of excitatory and inhibitory neurons, $\eta_i \sim \mathcal{N}(0.0, (\pi\tau_m)^2)$ the excitability parameter, $N = N_e + N_{hi} + N_{ai} = 100$, $N_e = 80$, $N_{hi} = 10$ and $N_{ai} = 10$ respectively the number of excitatory and Hebbian and anti-Hebbian inhibitory neurons, $g_e = 100$, $g_{hi} = 400$ and $g_{ai} = 200$ the global coupling strength for the excitatory neurons and Hebbian and anti-Hebbian inhibitory neurons. The coupling weights from neuron j to i is depicted by w_{ij}, t_j is the time of spike of the j-th neuron, and $\delta(t)$ is the Dirac delta function. Finally, $I_i(t)$ is the sensory input current and $\xi_i(t) \sim \mathcal{N}(0.0, (4\pi\tau_m)^2)$ is a Gaussian noise. We consider a fully connected network without self-connections.

We integrate the Eqs. 5.15, and5.16, using the Euler method. Whenever $V_i(t)$ reaches the peak value $V_p = 10$, the neuron i emits a spike (after a time of $\frac{1}{V_i}\tau_m$ seconds) and its membrane voltage is reset to $V_r = -10$ after a certain refractory period $\frac{2}{V_i}\tau_m$ seconds to compensate with the approximation $V_p = -V_r = 10$. These periods are equivalent to the theoretical time intervals to reach $V_i(t) = \infty$ from $V_i(t) = 10$ and to pass from $V_i(t) = -\infty$ to $V_i(t) = -10$ [116].

5.2.2 Plasticity functions

Regarding the adaptation of the weights w_{ij}, we use spike-timing-dependent plasticity (STDP) rules that depend on the time difference $\Delta t = t_i - t_j$ between the last spikes of the post-synaptic neuron i and pre-synaptic neuron j. The plasticity functions $\Lambda^+(\Delta t)$ and $\Lambda^-(\Delta t)$ from Eqs. 5.17 for potentiation and depression respectively, depend on the nature of the pre-synaptic neuron.

$$\Lambda^+(\Delta t) = \begin{cases} \Lambda(\Delta t), & \text{if } \Lambda(\Delta t) \geq 0, \\ 0, & \text{if } \Lambda(\Delta t) < 0, \end{cases} \qquad \Lambda^-(\Delta t) = \begin{cases} 0, & \text{if } \Lambda(\Delta t) \geq 0, \\ \Lambda(\Delta t), & \text{if } \Lambda(\Delta t) < 0. \end{cases} \tag{5.17}$$

For excitatory neurons we use a Hebbian STDP asymmetric function commonly used in the literature [117] described by Eq. 5.18.

$$\Lambda(\Delta t) = \begin{cases} A_+ e^{-\frac{\Delta t}{\tau_+}} - A_- e^{-\frac{4\Delta t}{\tau_+}} - f, & \text{for } \Delta t \geq 0, \\ A_+ e^{\frac{4\Delta t}{\tau_-}} - A_- e^{\frac{\Delta t}{\tau_-}} - f, & \text{for } \Delta t < 0, \end{cases} \tag{5.18}$$

with the time constants $\tau_+ = 0.02$s and $\tau_- = 0.05$s, the amplitudes $A_+ = 5.296$ and $A_- = 2.949$. The forgetting term $f = 0.1$ allows to have a constant small depression of the weights whatever the spike timing difference. It models the natural, constant and slow forgetting of memories [118].

For Hebbian (anti-Hebbian) inhibitory neurons we use a Hebbian (anti-Hebbian) STDP symmetric function [119, 120] described by Eq. 5.19.

$$\Lambda(\Delta t) = \pm A(1 - (\frac{\Delta t}{\tau})^2) e^{\frac{-\Delta t^2}{2\tau^2}} \mp f, \tag{5.19}$$

with time constant $\tau = 0.1$s, amplitude $A = 3$ and forgetting term $f = 0.1$.

5.2.3 Adaptation of synaptic weights

The evolution of the synaptic weights, which remain continually subject to adaptation, unlike more conventional learning systems, follows this ordinary differential equation:

$$\tau_l \dot{w}_{ij} = (-1)^{a_q}[\tanh(\lambda(w_q^l - w_{ij})) * \Lambda_q^+(\Delta t) + \tanh(\lambda(w_{ij} + w_q^u)) * \Lambda_q^-(\Delta t)] \qquad (5.20)$$

where q denotes if the pre-synaptic neuron is excitatory $q = e$ or Hebbian (anti-Hebbian) inhibitory $q = hi$ ($q = ai$), for excitatory (inhibitory) neurons we set $w_q^l = 1$ ($w_q^l = 0$) and $w_q^u = 0$ ($w_q^u = 1$), thus ensuring that the excitatory (inhibitory) couplings are defined in the following interval $w_{ij} \in [0 : 1]$ ($w_{ij} \in [-1 : 0]$). Moreover, $a_e = 2$ and $a_{hi} = a_{ai} = 1$, thus for inhibitory synapses, the plasticity functions $\Lambda^+(\Delta t)$ and $\Lambda^-(\Delta t)$ are inverted and multiplied by -1 since potentiation (depression) of inhibitory weights makes them converge towards -1 (0). Finally, $\tau_l = 0.2$s is the learning time scale for the adaptation.

5.3 Experiments and Results

5.3.1 Experimental Setup

A Khepera IV robot was placed on a table in the robotics laboratory. A human interacted with the robot, stimulating its IR sensors by inducing contact within the sensory field area with his hand (as shown in 5.3) in the following experimental conditions. Three repetitions of touch tests focusing on four metrics were carried out: Speed (Slow, Fast), Frequency (Low, High), Duration (Short, Long), and Intensity (Diffuse, Intense). For each metric, sensors underwent individual testing under each condition, resulting in a total of 64 tests (8 sensors x 4 metrics x 2 conditions). To reduce bias, both the order of conditions and the sensors were randomized. Each touch event was separated by 1 second, with sensor data captured at a frequency of 100Hz, ensuring a comprehensive dataset. The inclusion of randomization in sensor and condition order aimed to prevent any sequential bias and ensure the robustness of the experimental results. The experiment lasted 20 minutes, and data were collected throughout. The experiment was run 5 times, with similar results. Below we discuss a representative run.

5.3.2 Results

Nociceptors & Mechanoceptors output. In Fig. 5.3, we can observe the output of the nociceptive vector and of the physical information we used to compute it in a specific time windows between 125 and 150 seconds. Mechanoceptor vector has been computed as described in 2.2. These data are sent to the neuronal model.

Dynamics during learning. We first describe the dynamics of the network during learning of external sensory stimuli in Fig. 6.2 A. First, we observe that neurons associated with mechanoreceptors (excitatory neurons 64 to 127 and inhibitory neurons 144 to 159) respond to tactile input slightly earlier than neurons associated with nociceptors (excitatory neurons 0 to 63 and inhibitory neurons 128 to 143). This time delay between

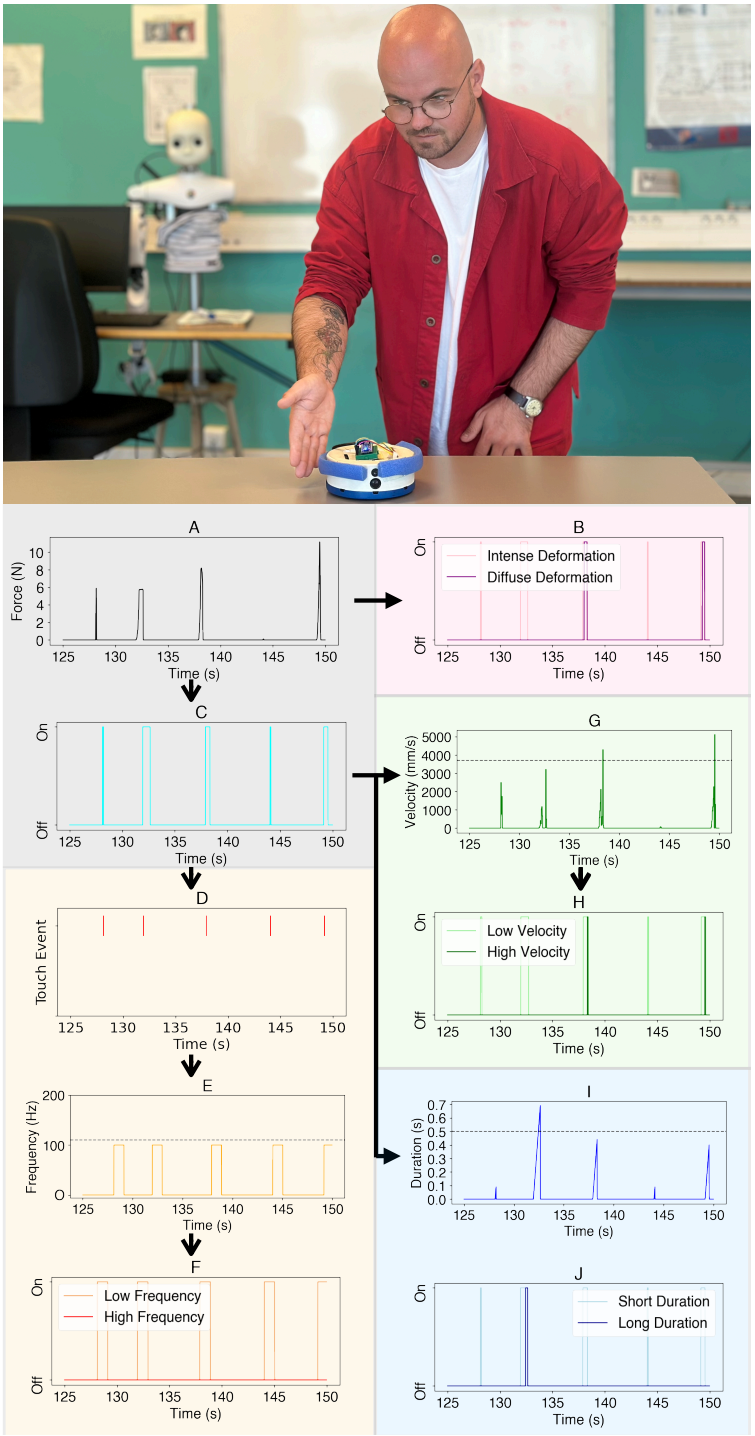


Figure 5.3: Picture of our experimental setup (top), and Dynamic Interactions and Time-Based Analysis of Tactile Sensory Events in a Nociceptive Model. Black arrows indicate the directional interactions between different tactile sensory events within the same time window. (A) Mean force applied to the nociceptive field over time. (B) Partitioning of deformation on the nociceptive blob over time. (C) Activation of touch on the blob over time, true if a certain amount of force is applied to the blob. (D) Touch event over time. (E) Frequency of touch in Hz over time. (F) Separation of frequency bands over time. (G) Velocity of touch in mm/s over time. (H) Segregation of velocity bands over time. (I) Duration of touch over time. (J) Division of duration bands over time.

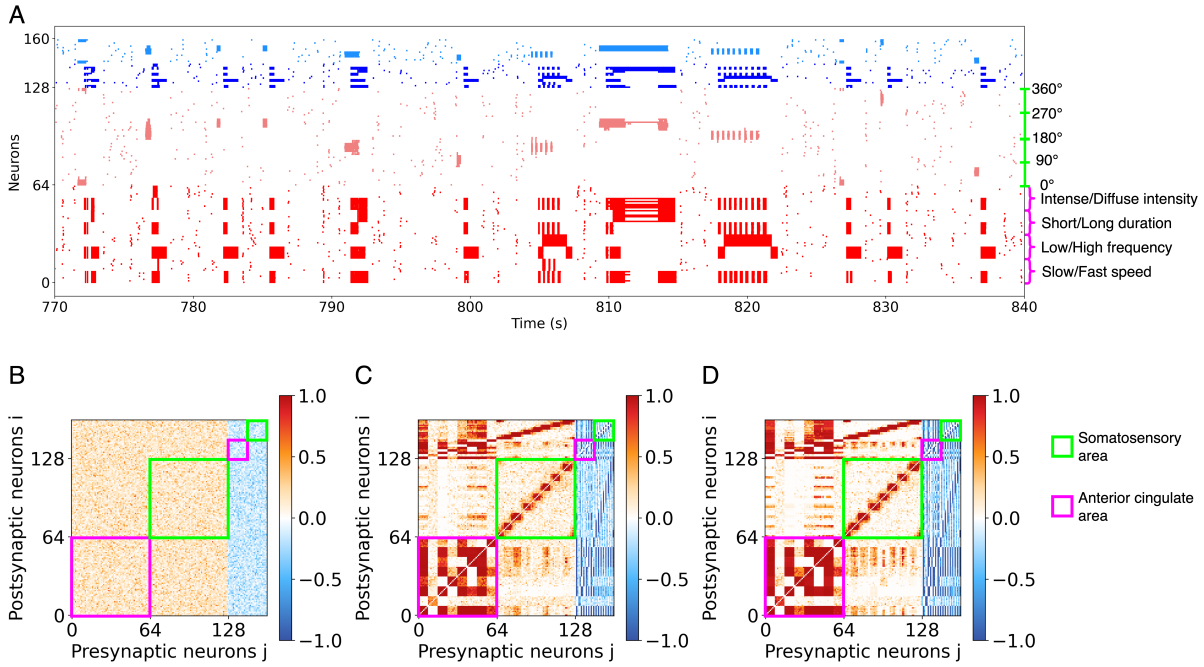


Figure 5.4: Neuronal simulation of touch stimulation. (A) The raster plot displays the firing times of excitatory (red dots) and inhibitory (blue dots) neurons during the simulation. Dark red (blue) dots represent spikes in the ACC (nociceptors), while light dots represent spikes in the somatosensory cortex (mechanoreceptors). (B,C,D) The matrices show the connection weights between neurons at the start, middle and end of the simulation. The color denotes if the connection is excitatory (red) or inhibitory (blue) or absent (white). The magenta area represents the ACC with touch features, while the green area represents the somatosensory cortex with locations of touch.

the information from the two types of sensor stems from the beta (for mechanoreceptors) and delta (for nociceptors) fibers, which transmit sensory information to the two cortical areas at different speeds.

In the somatosensory area (see light spikes), a touch on the robot is characterized by an increase in the firing rate of neurons associated with a specific location on the robot's body. Thus, a more spread contact will activate more neurons than a more targeted contact. In addition, a deeper touch will increase neuron activity more than a softer touch (i.e. higher firing of somatosensory neurons). We observe the same activation patterns in both excitatory and inhibitory populations, since they receive the same inputs.

On the side of the ACC area (see dark spikes), neurons increase their activity in the presence of particular touch features such as frequency bands, duration bands, deformation shapes and velocity bands. In this way, certain neurons are never activated at the same time, given the opposite nature of the features they encode (e.g. intense versus diffuse deformation). Conversely, certain features can be activated at the same time when they are not incompatible (e.g. a diffuse, long and low-frequency touch). In addition, it should be noted that some feature neurons activate only after other features have been activated. For example, a touch must be perceived as short before it can be considered long.

Resulting weight connectivity. This learning leads to the formation of particular structures in the weights connectivity of Fig. 6.2, B to D. Firstly, at the global level, we observe the formation of two modular structures, where excitatory neurons associated with the same sensory receptors (either nociceptors in magenta or mechanoreceptors in green) share strong connections, while connections between the two sensory areas are sparse and weak. This segregation between cortical areas is made possible by the beta and delta fibers described above, which prevent temporal correlations between information from nociceptors and mechanoreceptors, and hence their structural reinforcement.

In the area associated with mechanoreceptors (in green), we find that neurons associated with physically close sensors are strongly connected, while connections between distant areas are essentially suppressed. In other words, we obtain the formation of a kind of ring connectivity representing the robot's body.

Concerning the nociceptors area (in magenta), we can see that neurons coding for contradictory information (e.g. low and high frequencies) are totally decoupled. Nevertheless, some distinct feature neurons are strongly connected, showing that certain types of touch are characterized by different features. For example, a single touch may be perceived (by the robot or humans) as intense, long, with a low frequency and high speed. These joint activations will therefore couple the weight between these feature neurons. Moreover, we find that some feature neurons, such as those associated with low frequency touch, share connections with all other feature neurons. Indeed, these features almost always remain active, which also explains the few weak connections between the two cortical areas.

5.4 Discussion & Conclusion

In this chapter, we have presented a sensory body model for a mobile robot able to capture complex information about touch, including painful touch, from few data. We further investigated the coding of nociceptive features in an artificial neural model of ACC, a brain region associated with processing emotions and pain [121]. In the representation that emerged, we observed correlations and decorrelations between some nociceptive features extracted from different types of noxious stimuli, suggesting specific coding mechanisms.

Using a Khepera IV robot, we modeled a specific sensory body and tested various noxious stimuli designed to elicit distinct responses. The observed correlations and decorrelations between specific nociceptive inputs imply that some features co-activate, potentially encoding and discriminating different types of noxious stimuli. For example, in skin, pressure, scratch, and pinch stimuli activate distinct features. These observations support Acuña's [122] description of nociceptive coding in the ACC.

We also investigated how tactile information from nociceptors and mechanoreceptors can be learned by an artificial neural network to form two distinct areas, comparable to what can be observed in biology with the ACC and the somatosensory cortex. This highlights the segregation of information, with the specialization of brain regions for specific tasks or modalities [123]. These results echo those obtained by Bergoin et al. in [124, 113] with simpler stimuli.

Further, the ACC and somatosensory cortex individually provide information on the characteristics of the touch (what and how) and on its location (where). More precisely,

in our somatosensory network, we found that neurons coding for physically close robot body parts were more strongly connected than those for distant ones. This reminds us of the concept of semantic memory, where we find an association between mental representations and topology [123]. However one should keep in mind that here the network is all-to-all connected and the neurons have no topological organization.

Our model contributes to the explainability of robot behavior, since, given that the neural network is able to react to particular features and the location of touch, we could read these neural activities directly and associate them with particular behaviors or reflexes. In future work we could learn these associations and teach the robot to link certain features with types of pain or pleasure and particular movements. Finally, the neural network used would allow us to carry out experiments in more complex and changing environments, and assess the ability of the model to maintain continual learning without catastrophic forgetting of information encountered during exploration tasks.

Chapter 6

Influence of the social context and interaction on pain-modulated action selection

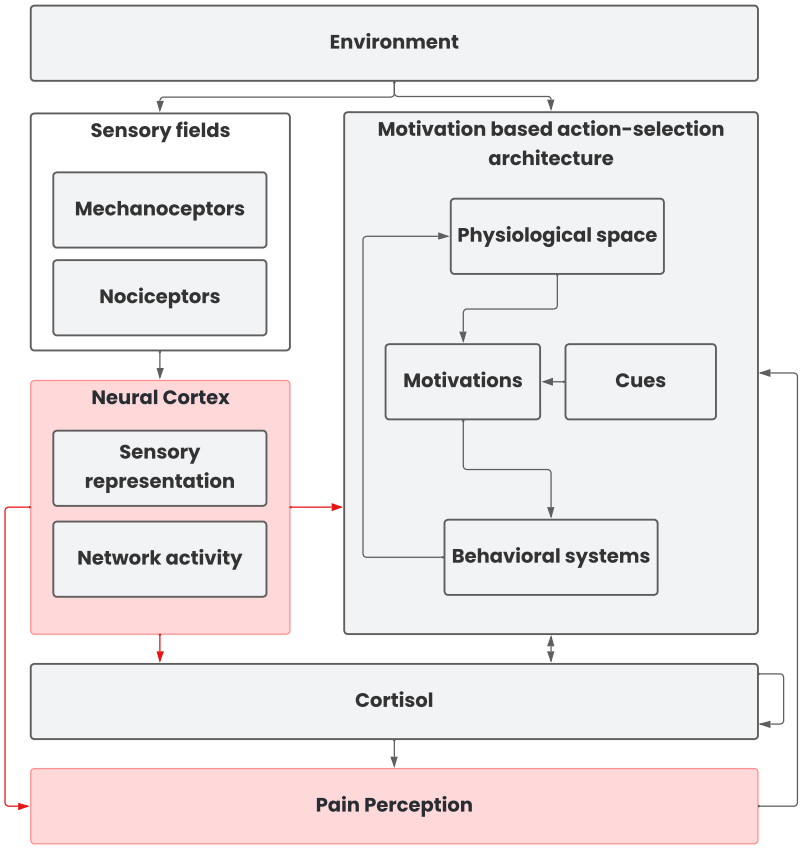


Figure 6.1: High-level diagram of our model and the effects we investigate. Red areas and arrows indicates the elements developed in this chapter.

Embodied representation plays a crucial role in pain perception. Body awareness and how we perceive our body significantly affect the pain experience in humans. The way the body

is represented, both mentally and physically, can alter the intensity and the perception of pain [125]. Distorted representations of body parts in chronic pain patients, such as those with fybromaliga, can lead to an increased sensation of pain [126]. Modifying these body representations through therapeutic methods like multi-sensory illusions (e.g. the rubber hand illusion) have shown to reduce pain perception by altering the brain's processing of body awareness. [127]. Thus pain perception shall not be exclusively a sensory experience but an embodied one, shaped by how the brain integrates the sensory inputs with cognitive and emotional states, highlighting the bidirectional relationship between body representation and pain perception [128].

Early life interactions, particularly sensory experiences, play a crucial role in the shaping of body representations [129, 130]. From childhood, multi-sensory inputs (e.g. touch, proprioception, vision) are integrated to form the basis of how individuals perceive and interact with their bodies and the external environments. Different early-life interactions, can result in differing developmental trajectories in how individuals perceive and experience their bodies.

In this chapter, I will propose a final action selection model that emphasize all of our previous work. Building on Chapter 4 cortisol modulated action selection architecture I will the sensory field representation and neural cortex architecture I presented in Chapter 5. I aim to study of different social interaction with predators will influence the body representation constructions and how it will affect the action selection mechanism in a robotic agent.

6.1 Model

The specific action selection model I use in this chapter builds upon the work described in Chapters 3 and 4. The agent, a Khepera-IV robot, will face a survival-related problem: it must consume three different resources in its environment to maintain internal homeostasis and survive. Various types and numbers of robotic agents (predators) will introduce survival-related dangers into the agent's environment. I will also incorporate the artificial mechanoception and nociception systems, along with the neural network described in Chapter 5, into our agent.

6.1.1 Action selection model

Our specific action selection model builds upon previous work on homeostatic homeostatically controlled motivation-based robot decision-making. In previous chapters, I presented specific elements of this architecture.

I add in this action selection a physiological variable to the physiological space linked to the notion of *physical integrity, and thus, a related motivation to* recover and a specific Behavioral system and a cue linked to the presence of another resource in the environment. This new dimension of the physiological homeostasis of the agent will add complexity to our agent's behavior and will force the robot to respond differently with damage induction. After being attacked by a predator, the robot will need to recover its physical integrity by going to a nest. This new challenge will increase the difficulty of maintaining other

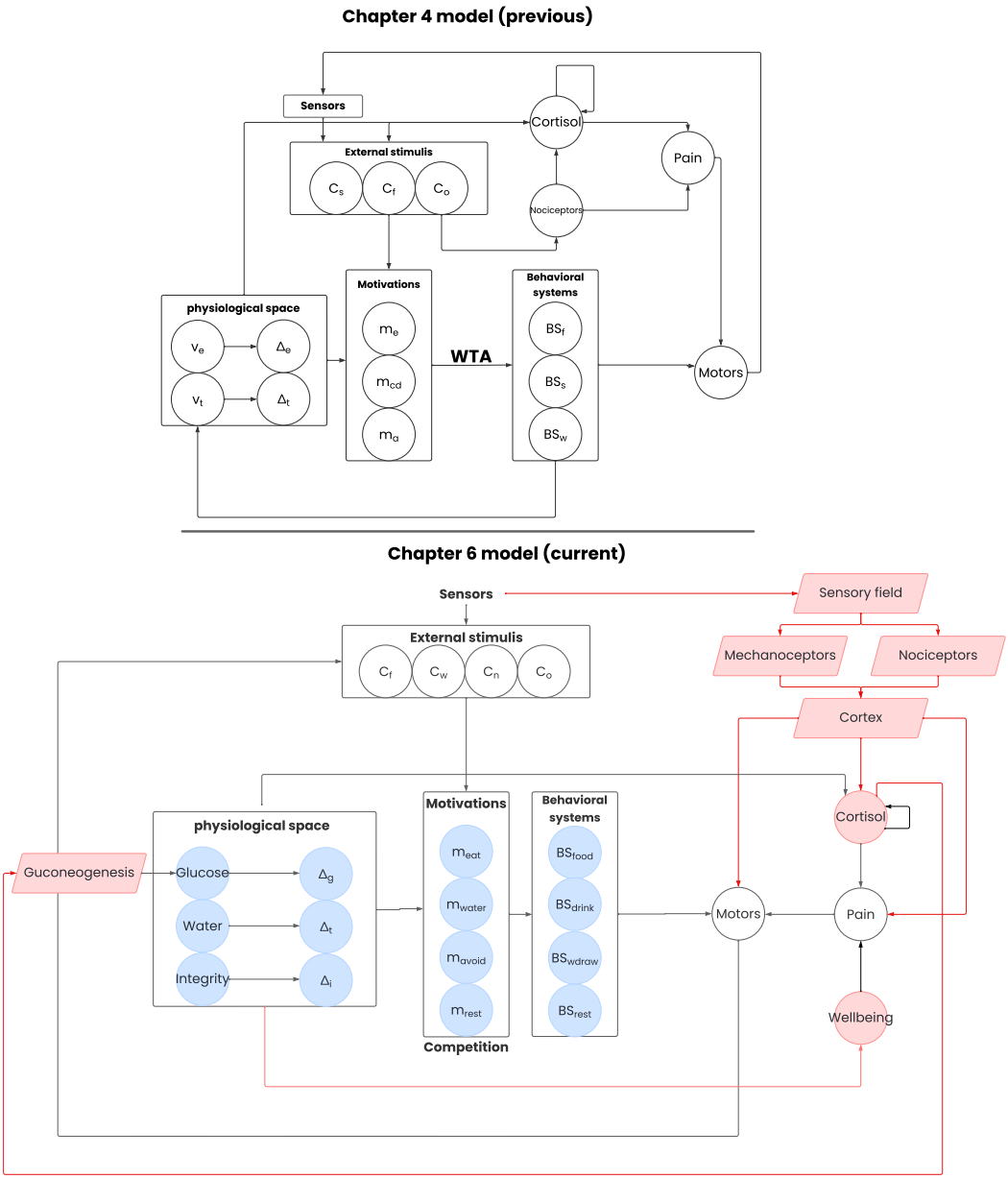


Figure 6.2: Granular view of the motivation-based action selection robot's model compared to the version presented in Chapter 4. Red elements and arrows are the new elements I added and blue elements are the specific elements modified without touching computation and logics. Names and elements are specified in Table 6.2

physiological variables within their viable ranges while trying to recover from damage. Elements of the architecture are presented in Table 6.2.

Elements described in previous chapters that need to be precisely detailed in the following sections are used as stated in the previous chapters.

Elements	Name
Physiological variables (v)	glucose, water, integrity
Sensors (internal & external)	Food, Water source, Nests, Ground I.R. Sensors, Nociceptors, Mechanoceptors
External stimuli, or cues (c)	"water," "food," "nest," obstacles
Motivations (m)	eat, drink, recover, avoid
Behavioral systems	Consume food, Drink, Rest, withdraw

Table 6.1: Elements of the action selection architecture

Behavioral System	motivation	Behavior	Effects
Consume food	eat	consume food	↑ glucose, ↓ water
		seek for food	↓ glucose, ↓ water
		wander	↓ glucose, ↓ water
Drink	drink	consume water	↓ glucose, ↑ water
		seek for water	↓ glucose, ↓ water
		wander	↓ glucose, ↓ water
Rest	recover	rest in nest	↑ integrity, ↓ energy, ↓ water
		seek for nest	↓ glucose, ↓ water
		wander	↓ glucose, ↓ water
Withdraw	avoid	withdraw	↓ glucose, ↓ water

Table 6.2: Behavioral systems, their linked motivations, behaviors, and effects. ↑ indicates that the behavior increase the level of a physiological variable, ↓ that it decrease it level.

6.1.2 Internal states and artificial hormone

Our action selection model is modulated, like the previously described model, by various internal states and artificial hormones. These values represent the robot's perception of its internal state and its external environment based on past states, physiology, and external stimuli.

6.1.3 Wellbeing

In the context of our homeostatically driven action selection model, wellbeing can be understood as the hedonic satisfaction of homeostatic needs or the reduction of physiological error (i.e., through resource consumption). The computation has been adapted from the previous chapter's description to give greater weight to the most significant deficits.

$$\begin{aligned} Err &= \Sigma_i(phys.err_i) \\ MaxErr &= max(phys.err_1, ..., phys.err_i) \\ \text{wellbeing} &= 1 - \frac{\text{Err}}{\text{MaxErr}} \end{aligned} \tag{6.1}$$

6.1.4 Artificial (simulated) Cortisol

Building on the artificial cortisol model described in Chapter 4, we have implemented an artificial cortisol hormone in our system. This hormone is designed to suppress nonessential functions during fight-or-flight situations and modulates the robot's perception of damage.

As outlined in Chapter 5, the robot's damage perception is based on sensory input from mechanoreceptors and nociceptors.

The artificial hormone is released by a gland influenced by three factors: wellbeing, pain perception, and external stimuli.

Stimuli are calculated using both mechanoreceptive and nociceptive inputs. Specifically, the damage perception is computed as follows:

$$\begin{aligned}\text{damage} = \sum_{i=1}^{3} \max_i(\{\text{mechanoceptor}_1, \text{mechanoceptor}_2, \ldots, \text{mechanoceptor}_n\}) \\ +\text{nociceptive\_activation}\end{aligned} \tag{6.2}$$

Here, nociceptive activation is calculated as the mean of the activations of all nociceptive sensors.

Hormone release rate is computed as combination of damage perception modulated by robot internal state (pain and wellbeing levels). As seen in Chapter 5 hormone release rate is also modulated by previous hormone concentration.

$$\begin{aligned}\text{release\_rate} = \mathit{damage} \cdot (1 + \gamma \cdot c_{\text{cortisol}}(t-1)) \\ +\alpha \cdot \text{pain\_level} + \beta \cdot (1.0 - \text{wellbeing\_level})\end{aligned} \tag{6.3}$$

Where γ represents the influence of the previous cortisol concentration, α and β are the respective influence of pain and wellbeing perceptions, as in previous chapters, cortisol concentration is computed using release rate and a constant decay.

6.1.5 Gluconeogenesis

Gluconeogenesis [45] is the metabolic pathway that results in the synthesis of glucose from cortisol. It is the process by which blood sugar levels can be maintained at a satisfactory level when carbohydrate intake is reduced. In many animals, it can occur during intense exercise or fasting periods. It is a valuable tool for maintaining energy homeostasis.

In this model, cortisol is a hormone influenced by both external danger and the internal state of the robot. It primarily appears when the robot lacks resources or is subjected to high levels of stress-induced danger that prevent it from seeking resource consumption. Cortisol enables the robot to physiologically maintain its glucose levels and seek the energy necessary for survival during intense danger phases.

We use this pathway in our model by modulating the physiological variable representing glucose with artificial cortisol concentration as follows.

$$glucose = glucose(1 + \alpha * cortisol_{concentration}) \tag{6.4}$$

6.1.6 Cues

In our setup, we now use colored tiles as resources instead of different textures. These tiles are detected as cues by the robot via an RGB sensor. To calculate the strength of a sensation, we calibrate the RGB sensor for each experiment. Using the reference values of the tiles, we can calculate the robot's cue. To maximize the precision of the sensation, we also use the robot's ground I.R. sensor to verify not only the RGB sensor's reading but also whether a resource is actually detected. This method helps us avoid false positives that could occur in previous versions of our model by comparing two perceptions to compute the cue associated with a resource.

Colors can be viewed as points in a 3-dimensional RGB space (Red, Green, Blue). To calculate the distance between two points, we can use the Euclidean distance:

$$\text{distance} = \sqrt{(R_1 - R_2)^2 + (G_1 - G_2)^2 + (B_1 - B_2)^2} \tag{6.5}$$

Using the distances between our reference colors and a "neutral" ground color, we can compute the cue as follows:

$$c_{\text{level}} = \left(1 - \frac{\text{distance}}{\text{refDist}}\right) + \alpha \cdot \left(\left(1 - \frac{\text{distance}}{\text{refDist}}\right) \cdot \text{salience}\right) \tag{6.6}$$

Where **refDist** is the distance between the current RGB sensor reading and the reference value, and **distance** is the distance between the current RGB sensor reading and the reference value of the resource associated with the cue. **Salience** refers to the cortisol level, which, when the robot is under stress, enhances its perception of the resource or the danger.

6.1.7 Sensory field navigation

Building on the concept of the sensory field we previously developed, we propose a novel navigation algorithm for obstacle avoidance and wandering. This algorithm aims to minimize the deformation of the sensory field, maintaining it as close as possible to its original state. Doing so provides more precise movements than Braitenberg behavior, leading to a more accurate navigation strategy that reduces unnecessary actions and conserves energy, particularly in fight-or-flight situations. We can compare this approach to an attempt by the robot to maintain a peripersonal space that is clear of obstacles.

The first step in this approach is to compute the centroid of the deformed sensory field. When the sensory field is undisturbed, the centroid represents its center. However, in the presence of deformation, the centroid shifts to reflect the center of the deformation.

$$C_x = \frac{1}{n}\sum_{i=1}^{n} x_i, \quad C_y = \frac{1}{n}\sum_{i=1}^{n} y_i \tag{6.7}$$

Once we get the centroid, we can compare its position to the robot's "head." As our robotic platform is circular, we choose to compute the angular difference between the centroid and the head.

$$\begin{aligned} \theta_{\text{centroid}} &= \text{atan2}(y_{\text{centroid}}, x_{\text{centroid}}) \\ \theta_{\text{head}} &= \frac{\pi}{2} \\ \text{error} &= \text{atan2}\left(\sin(\theta_{\text{centroid}} - \theta_{\text{head}}), \cos(\theta_{\text{centroid}} - \theta_{\text{head}})\right) \end{aligned} \tag{6.8}$$

Once we get this error, we can send a motor command that will reduce the error and prevent the deformation of the sensory field.

6.1.8 Pain perception & neural cortex

Pain perception is constructed both from sensations related to the robot's internal states and is modulated by artificial cortisol. It also relies on the activity of the simulated Anterior Cingulate Cortex.

In fact, our model operates on two different time scales, each functioning at separate speeds. The action selection model runs at a frequency of 25 Hz, while the cortex activity and the activation of spike neurons run at a frequency of 100 Hz.

Therefore, our model accumulates the neurons that activate between each iteration of the action selection process, and we use this accumulated cortical activity to calculate the robot's pain perception. The computation of pain perception is done as follows:

We compute the mechanoreceptive activity, denoted as *mechano_acc*, by considering the activity levels across all mechanoreceptive spikes. Each spike neuron registers activation events based on neural context. To compute *mechano_acc*, we sum the activation events for all spike neurons and then divide this sum by the total number of spike neurons. This provides an average level of mechanoreceptive activity, which reflects how active the mechanoreceptive system is in response to external stimuli. This is expressed as:

$$\text{mechano\_acc} = \frac{\sum_{i=1}^{n} \text{activation\_events}_i}{n} \tag{6.9}$$

We compute using the same method the nociceptive accumulated activity denoted as *noci_acc*. Using both these factors and internal state and artificial cortisol concentration, we can compute robot pain perception as follows :

$$pain = (\alpha \cdot mechano\_acc + \beta \cdot noci\_acc) * (1 - \psi \cdot wellbeing + \gamma \cdot cortisol)) \quad (6.10)$$

Where α and β adjust the respective effects of mechanoceptive and nociceptive activity on pain perception, determining where and how the robot is touched. Wellbeing decreases pain perception, while cortisol increases it.

6.2 Experimental setup & conditions

In this section, we present the results of two series of experiments designed to evaluate the relevance of using a neural cortex that creates a memory of sensation in the context of our survival-related problem. We will evaluate the robot in environments where it is subjected to influence and interaction in a social context with different types and numbers of predators, aiming to disrupt its goal-achieving behaviors.

Our hypothesis is that in the various scenarios to which we will expose the robot, the greater the danger, the more effectively the sensory memory will form in the cortex, and the more relevant this memory will be in responding to the homeostatic survival challenges.

By adding a new dimension to our model and moving from a Two-Resource Problem to a Three-Resource Problem, we have also increased the difficulty of the task. This allows us to observe how, in stressful and dangerous environments, the robot will choose to act to survive. In this chapter, the goal is not to evaluate whether the robot will survive but how it will respond to the challenge. However, the survival rate can be an interesting metric.

As in the previous chapters, we have embedded our architecture into a Khepera IV robot, but this time, we will use a greater variety of predator types to assess our robot's performance.

The environment (Fig. 6.3) consists of a 2m by 2m wooden arena containing three types of resources represented by floor tiles of different colors, which the robot can detect and discriminate between using RGB sensors.

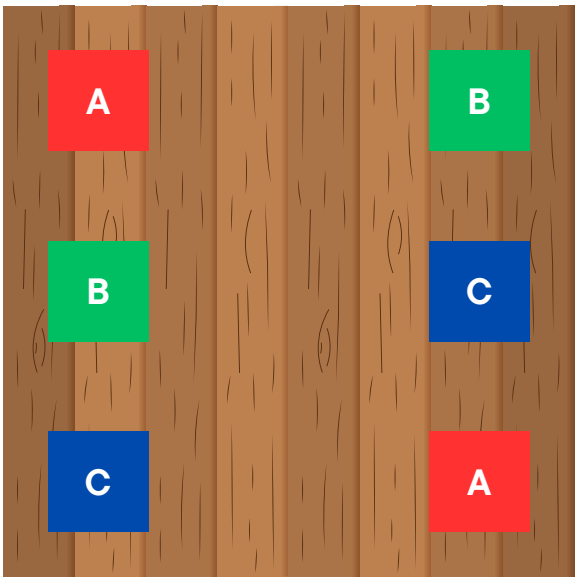


Figure 6.3: 2m by 2m wooden arena with color resources (A) food, (B) nest and (C) water

6.2.1 Predators

In order to maintain environmental variability and stress-related danger, as in previous experiments, we will introduce different types of predators. Some will be Thymio-II robots exhibiting predatory behaviors, while the experimenter will manually induce others. We aim to test different conditions under various types of stress-related danger.

On the one hand, building upon previous Thymio-II predators, we implemented two types of behaviors: **Stalking** and **Hunting**. These two behaviors are inspired by Braitenberg's ideas. **Stalking predators** slowly wander around the environment, avoiding walls and edges. When they encounter a moving object, they follow it and attempt to inflict damage by attacking at high speed, inducing a circular-type damage pattern to maximize the deformation of the sensory field caused by the Thymio-II. **Hunting predators** actively search for moving objects in the environment, and when encountered, they attack in the same manner as stalking predators.

On the other hand, we use the experimenter to introduce two other types of predation. Our goal is to observe how the formation of sensory memory can impact our model in different social contexts, so we propose to compare early life experiences with continuous life experiences.

First, we propose an **Early Life predation** scenario, where damage is manually induced by the experimenter every 3 seconds during the first third of the experiment. The robot is left to explore the environment, and damage is induced during the last sixth of the experiment. This scenario can be compared to a **Constant predator**, where the experimenter induces damage every 3 seconds throughout the entire experiment.

6.2.2 Experiment parameters

We conducted two series of experiments to evaluate the effects of different numbers and types of predators. Each experimental condition was run 5 times, resulting in a total of 150 runs, which will be discussed in the following sections.

6.2.3 Number of predators

This first set of experiments compares the relevance of our model with different numbers of Thymio-II predators, with the two types of behaviors that we implemented, with or without the cortex activity. All these runs were set to last 300s, even if, in some cases, the robot did not manage its physiological space within the permitted range, which led to the death of the robot. Different conditions tested are detailed in Table 6.3.

Predator Type	**Number of Predators**	**Cortex Memory**
Hunter	0	Yes
Hunter	0	No
Hunter	1	Yes
Hunter	1	No
Hunter	2	Yes
Hunter	2	No
Hunter	3	Yes
Hunter	3	No
Hunter	4	Yes
Hunter	4	No
Stalking	0	Yes
Stalking	0	No
Stalking	1	Yes
Stalking	1	No
Stalking	2	Yes
Stalking	2	No
Stalking	3	Yes
Stalking	3	No
Stalking	4	Yes
Stalking	4	No

Table 6.3: Experiment 1 - Duration 5 min

6.2.4 Type of predators

We conducted a second set of experiments to evaluate how our agent responded to different types of stress-related predation. In this set, the runs were extended to evaluate the effects of various predation types better, particularly to assess the influence of early-life predation during a more extended period without damage stimuli. We compared these conditions to previously tested predation types using both types 2 Thymio-II. Each experiment was set to last 900 seconds for each condition, even in cases where the robot

failed to maintain internal homeostasis and "died." Different conditions tested are listed in Table 6.4.

Predator Type	**Number of Predators**	**Cortex Memory**
Hunter	2	Yes
Hunter	2	No
Stalking	2	Yes
Stalking	2	No
Constant	N.A.	Yes
Constant	N.A.	No
Early Life	N.A.	Yes
Early Life	N.A.	No
No Predator	0	Yes
No Predator	0	No

Table 6.4: Experiment 2 - Duration 15 min

6.2.5 Live interface

As previously explained, we have an action selection model and a neural cortex running at different frequencies. The action selection model runs directly on the Khepera-IV, while the neural cortex operates on a 2022 M2 MacBook Air. Both systems communicate via a TCP NODELAY interface we implemented to maximize transmission speed. We also developed a web interface to monitor the experiments, as shown in Fig. 6.4.

This web interface is implemented using a Node.js server that captures communications between the Mac and the Khepera and displays information using a JavaScript library.

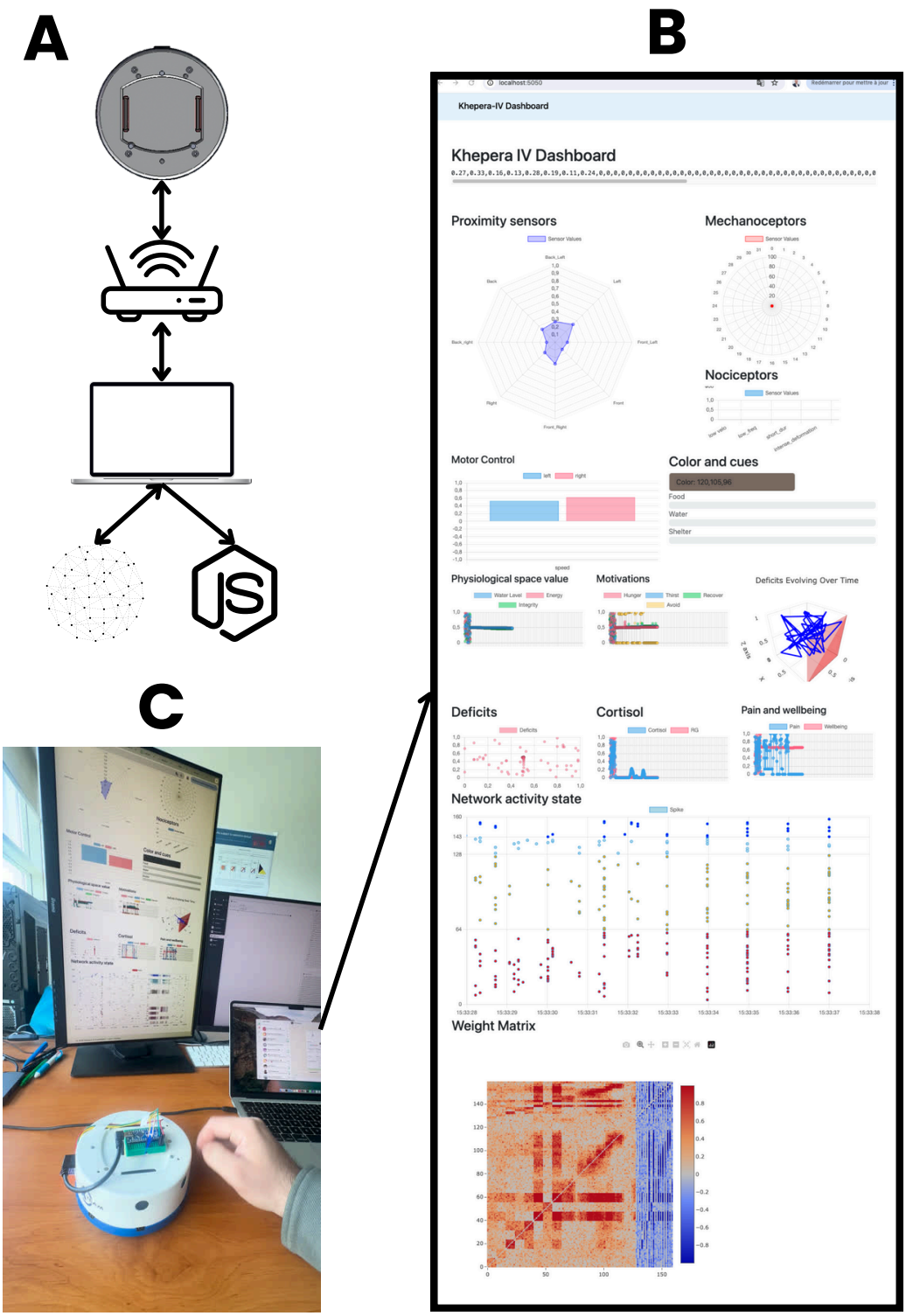


Figure 6.4: Graphical representation and representation of the live interface we developed. (A) summarize graphically the global architecture of the model. Khepera communicates to macbook via a rooter. Macbook that communicates back and forth to the neural cortex and to the Khepera and that sens information to the Node.js server and webpage to display. (B) is a screenshot of the webpage where we can display informations about the model such as live raster plot and weight connectivity. (C) is a picture of the live setup.

6.3 Results

In this section we will observe and discuss some of results of the conducted experiments.

6.3.1 Viability of the model

As previously mentioned, the two series of experiments differed in duration. However, in both series, we can observe that under "extreme conditions," the survival rate varies depending on the presence of cortex activity.

Firstly, as shown in Fig. 6.5, we observe that when there are fewer than or equal to two predators (as described in Chapter 4), both with and without cortex activity, the survival rate is 100%, which is consistent with previous findings. However, with three stalking predators, the survival rate drops to 80% without cortex activity, while it remains 100% with cortex activity. Similarly, with four predators, we see a significant difference in survival rates: 80% vs. 100% with stalking predators, and 60% vs. 40% with hunting predators, depending on the presence of cortex activity.

These results suggest an improvement in viability when cortex activity is present, as the formation of sensory memory appears to enhance the robot's ability to adapt to stress-related dangers.

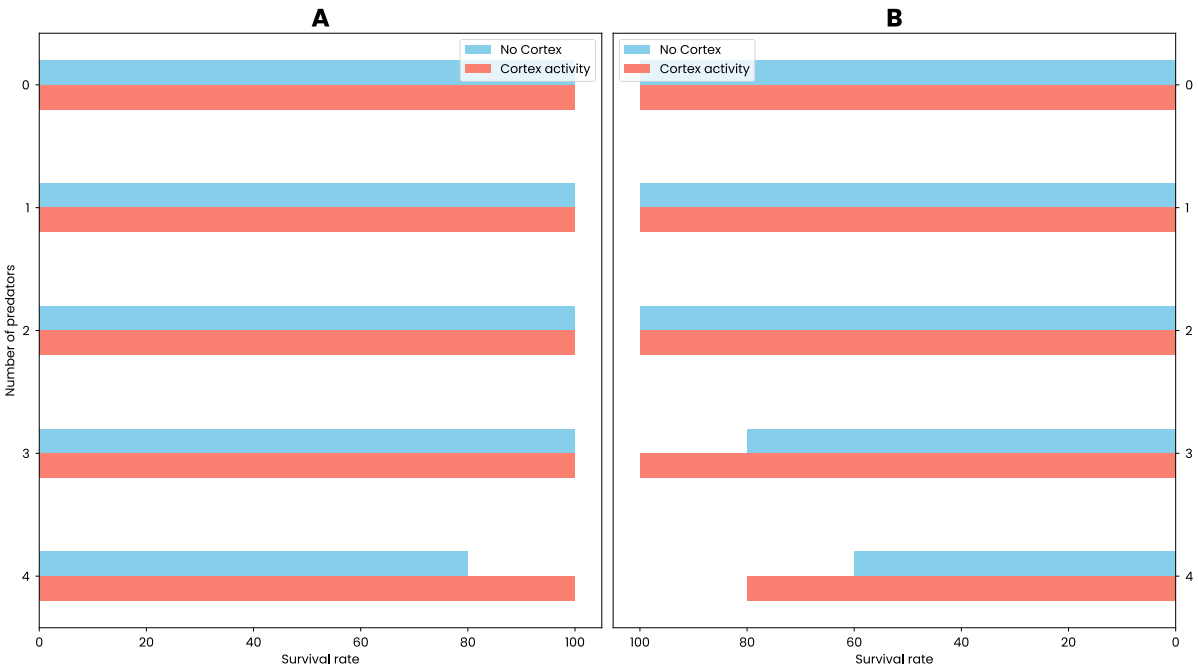


Figure 6.5: Survival rate of our model after 300s with (A) Stalking predators and (B) Hunting predators

Secondly, as shown in Fig. 6.6, we observe that viability also varies depending on the type of predation the robot encounters; with both Hunting and Stalking predators, even in the more extended experiments, the survival rate remains as high as 100%. However,

when subjected to Early Life and Constant predation, a significant difference in lifespan is observed.

In the case of Early Life predation, the survival rate is slightly lower, with 60% survival without cortex activity and 80% with cortex activity. The most notable difference is seen with Constant predation, where only 40% of the robots survived without cortex, compared to 80% with cortex.

This highlights the importance of embodied representation in extreme stress-related danger. In this social context of predation, the formation of sensory memory with cortex activity significantly helped the robot maintain internal homeostasis, improving its chances of survival.

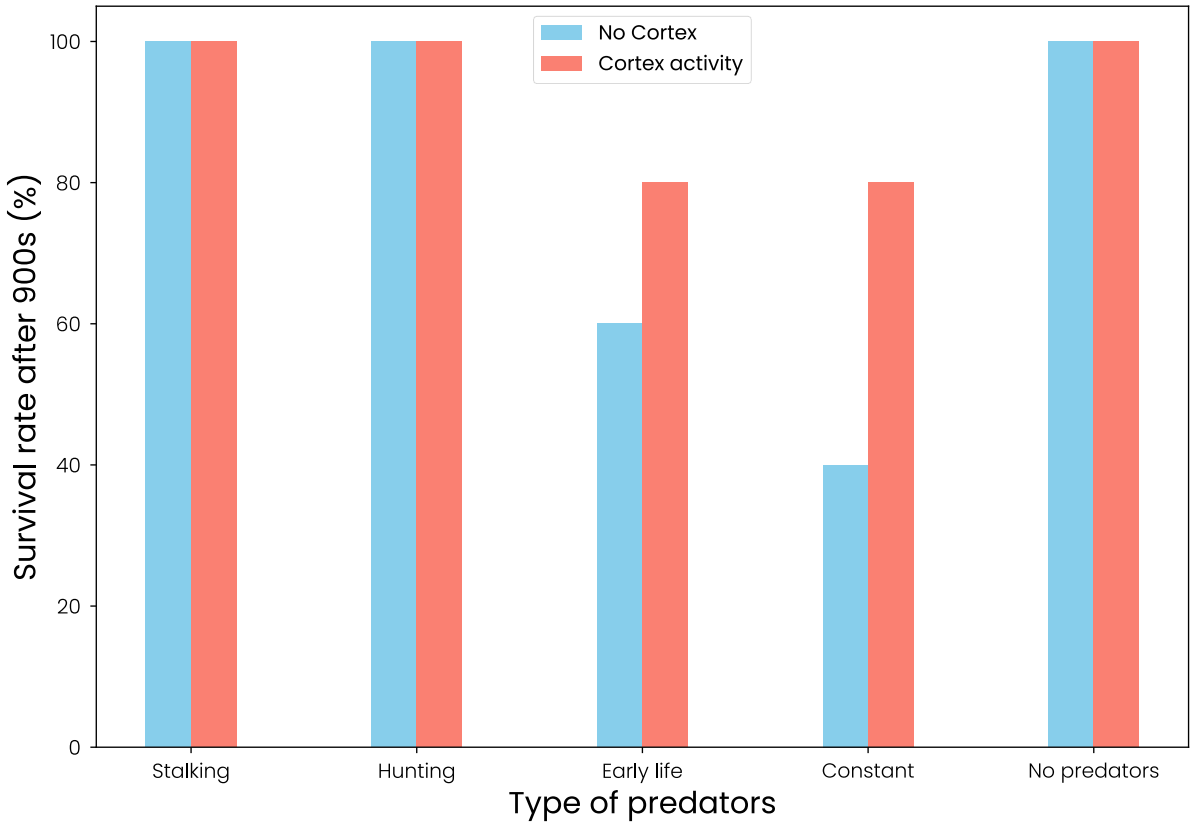


Figure 6.6: Survival rate of our model after 900s with different type of stress-related predation

In the following sections we will discuss specific runs and highlight some metrics we found relevant to assess model viability.

6.3.2 Dynamics during learning

We can describe the network dynamics during the learning of external sensory stimuli for a specific run shown in Fig. 6.7. The observed run corresponds to the Early Life

predator scenario within the time window when noxious stimuli were no longer applied to the robot, between 4 and 6 minutes.

In this time frame, we observe that neurons associated with mechanoreceptors (excitatory neurons 64 to 127 and inhibitory neurons 144 to 159) respond to tactile input slightly earlier than neurons associated with nociceptors (excitatory neurons 0 to 63 and inhibitory neurons 128 to 143).

Somatosensory neurons (lighter spikes associated with mechanoreceptors) fire based on the location of the touch, while neurons in the Anterior Cingulate Cortex (darker spikes associated with nociceptors) fire depending on the type of stimulus. In this live experiment, we can observe that different nociceptive features are activated in the ACC, and not all features fire simultaneously.

Some somatosensory neurons tend to fire more frequently than others, which can be explained by the navigation algorithm. The robot tends to face multiple objects with its head and less so with other parts of its body. After the neurons associated with the robot's head fire, we observe that other locations fire continuously in a circular pattern, indicating that the robot turns to avoid obstacles.

Additionally, we can observe an empty phase after the experimenter stops touching the robot for around 4.75 minutes, during which neurons fire randomly. This suggests memory recall by the network and highlights that the sensory representation has been learned by this point.

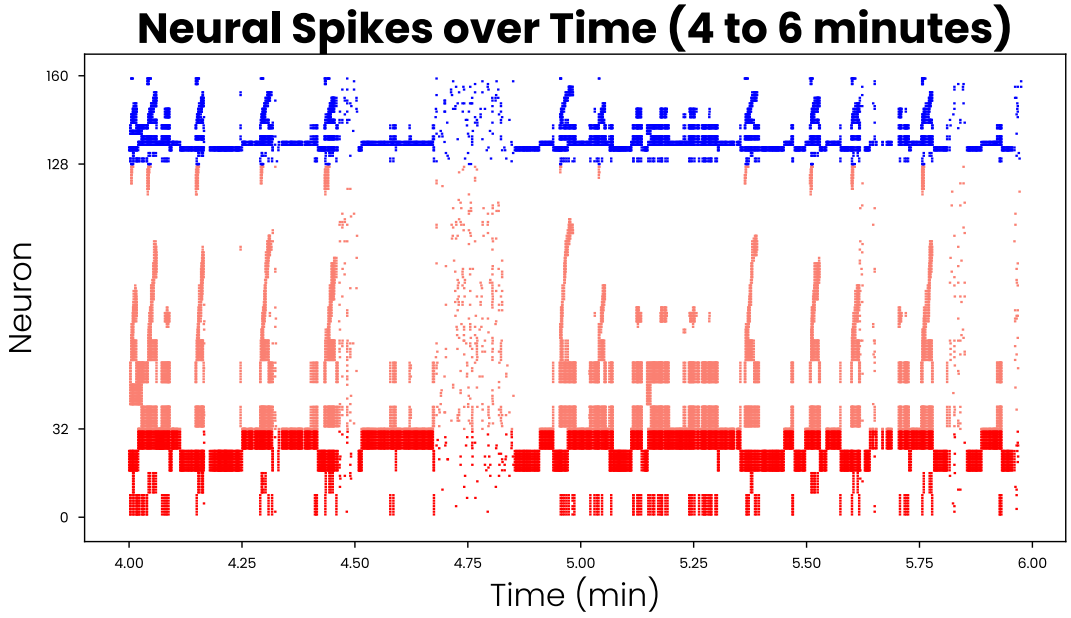


Figure 6.7: The raster plot displays the firing times of excitatory (red dots) and inhibitory (blue dots) neurons during the simulation. Dark red (blue) dots represent spikes in the ACC (nociceptors), while light dots represent spikes in the somatosensory cortex (mechanoreceptors)

6.3.3 Resulting weight connectivity

We observed network activity for the early-life predator between 4 and 6 minutes. Now, we will examine the evolution of the resulting weight connectivity in the same scenario. As shown in Fig. 6.8, learning progresses clearly during the first 5 minutes, becomes more sporadic during the period without noxious stimuli, and strengthens with the return of the negative signal.

This learning again reveals two distinct structures in the weight connectivity, representing the Anterior Cingulate Cortex and the Somatosensory Cortex. This segregation is once again made possible by the difference in the transmission of nociceptive and mechanoreceptive signals, thanks to the beta and delta fibers mechanisms described in Chapter 5. However, the two structures appear more strongly correlated than in the previous chapter. This is due to the change in stimuli during the learning phase. In Chapter 5, a rigorous experimental protocol ensured that the model was stimulated at all points in various ways, with separation delays between each touch. In our model, touch is induced more naturally as the robot also navigates its environment and encounters obstacles, such as the barrier closing off the arena.

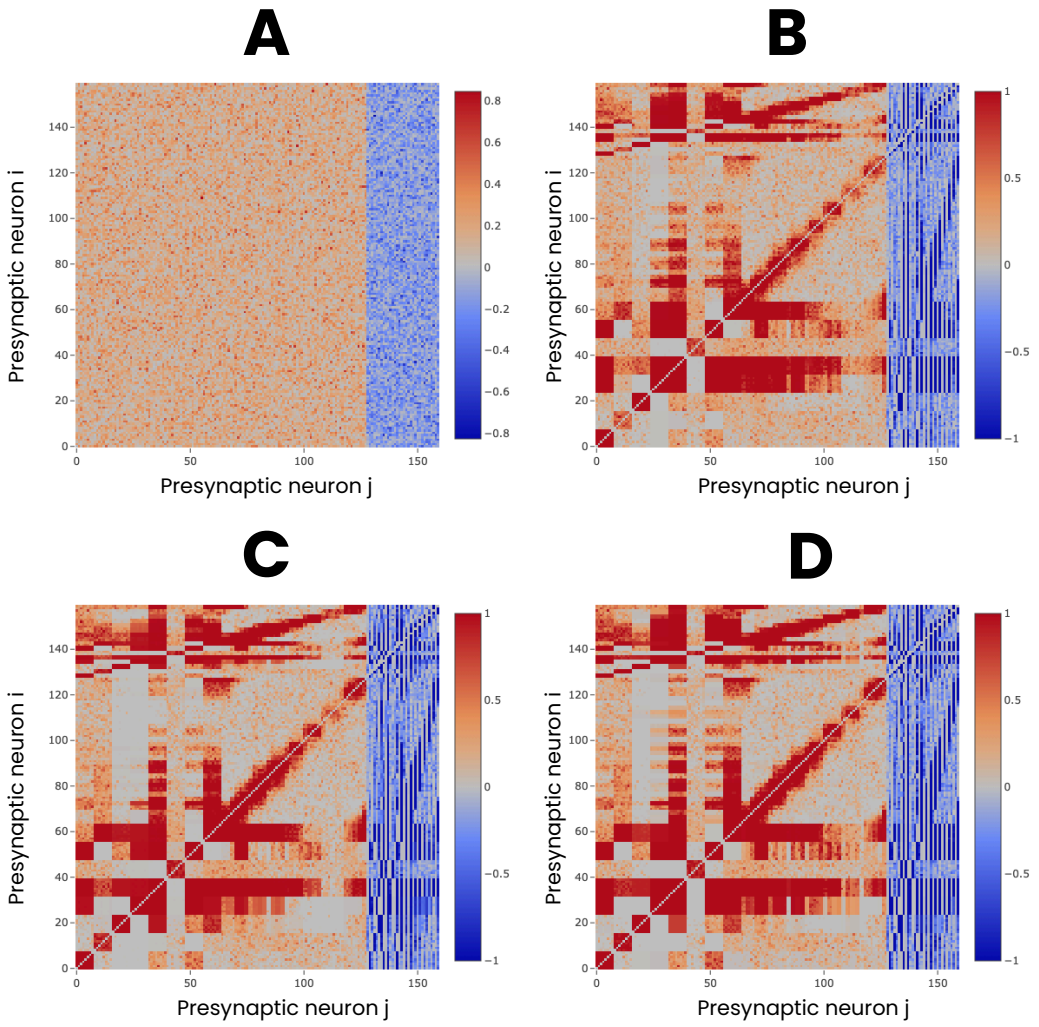


Figure 6.8: The matrices show the connection weights between neurons at (A) 0 min, (B) 5 min, (C) 12 min, (D) 15 min. The color denotes if the connection is excitatory (red) or inhibitory (blue) or absent (white)

Let us examine the resulting weight connectivity in more detail across different scenarios. We can observe that different social interaction contexts, with various types of predators and different levels of stress-related danger, lead to more or less defined and segregated connectivity matrices.

For instance, in Fig. 6.9, in a scenario without predation, we observe at the end of the 5-minute experiment that the Somatosensory Cortex is weakly defined and that the Anterior Cingulate Cortex only highlights a few nociceptor features. In this particular context, the only interactions that deform the sensory field and, therefore, activate the mechanoreceptors and nociceptors are the interactions with the arena's edges. Suppose we examine Fig. 6.10, representing the same scenario but with a 15-minute duration. We see that the Somatosensory Cortex becomes more defined, but the neurons associated with the back of the robot are not defined at all nor connected to the rest of the cortex. Indeed, without stimulation, this cortex area remained inactive, resulting in only a partial construction of the robot's sensory representation.

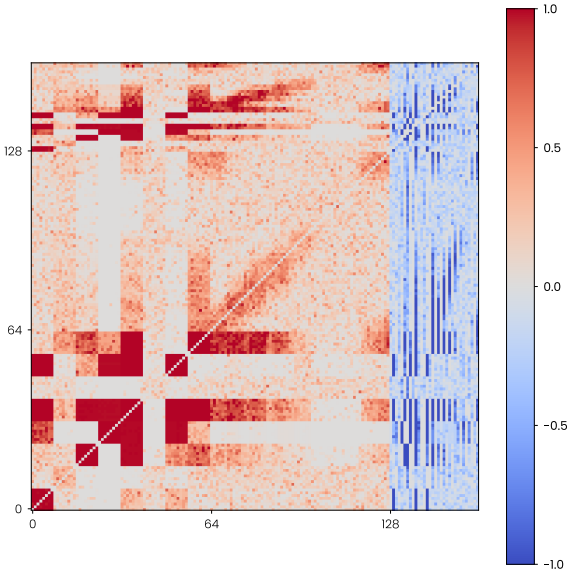


Figure 6.9: The matrices show the connection weights between neurons at the end of the simulation. The color denotes if the connection is excitatory (red), inhibitory (blue), or absent (white) after 5 minutes of experiment with 0 predators.

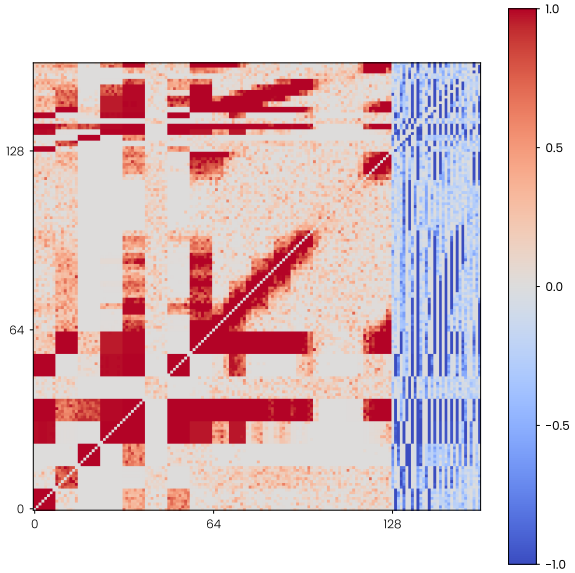


Figure 6.10: The matrices show the connection weights between neurons at the end of the simulation. The color denotes if the connection is excitatory (red), inhibitory (blue), or absent (white) after 15 minutes of experiment with 0 predators.

We can also observe in Fig. 6.16 and Fig. 6.17 that in two scenarios with high levels of stress-related predation, but with two different types of predators (Stalking and Hunting, respectively), we obtain two distinct connectivity matrices with varying degrees of definition. Indeed, the Somatosensory Cortex and the Anterior Cingulate Cortex are much more correlated with the Hunting predators. This is due to less distinct stimuli caused by the large number of predators, which repeatedly induce noxious stimuli in our agent, diminishing the effect of the delta and beta fibers.

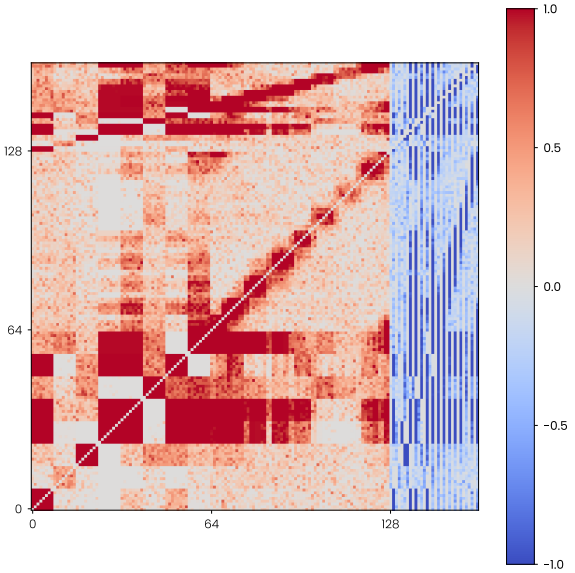


Figure 6.11: The matrices show the connection weights between neurons at the end of the simulation. The color denotes if the connection is excitatory (red), inhibitory (blue), or absent (white) after 5 minutes of experiment with 4 Stalking predators

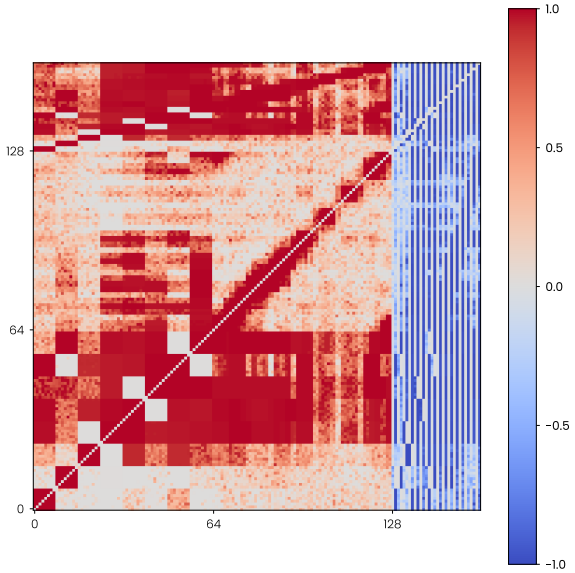


Figure 6.12: The matrices show the connection weights between neurons at the end of the simulation. The color denotes if the connection is excitatory (red), inhibitory (blue), or absent (white) after 5 minutes of experiment with 4 Hunting predators

6.3.4 Intensity of cortisol over time

If we observe cortisol intensity over time, we can see in Fig. 6.13 (top) and (bottom) that in a high-stress scenario (i.e., with many predators), the cortisol level gradually increases throughout the experiment. This demonstrates the phenomenon and mechanism of hysteresis, highlighting that in a scenario where a high-stress level is induced, cortisol levels, influenced by previous states, continue to rise without significant reduction during the run. It is worth noting that the increase is more consistent and smooth in the scenario where the cortex is activated.

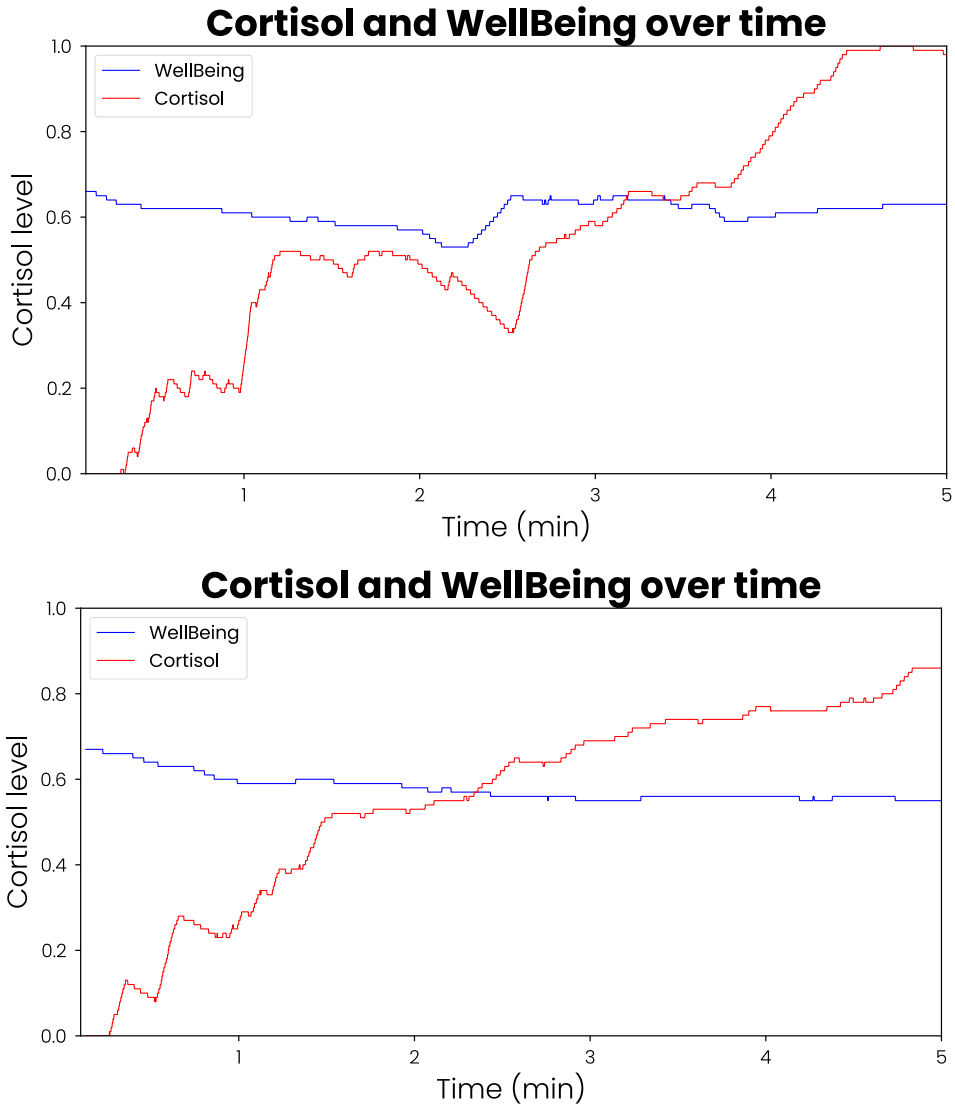


Figure 6.13: Intensity of Cortisol and Wellbeing over Time with four stalking predators: No Cortex (top) and Cortex activity (bottom)

These results suggest that the construction of sensory representations tends to enhance sensitization and smooth the mechanisms that increase cortisol levels. However, as we will observe in the second set of experiments, this observation becomes more nuanced. After the stage of constructing the sensory representation, another mechanism appears to emerge.

Indeed, as shown in Fig. 6.14 in the context of Early Life predation, we can observe two radically different patterns in cortisol intensity. In Fig. 6.14 (A), where the cortex is active, we see that after the predation phase, the sensory representation of the robot is relatively established. The cortisol intensity decreases after the predation stimulus ends at 5 minutes and only rises again when the stimulus is reintroduced at the end of the experiment, at 12 minutes 30. In contrast, in Fig. 6.14 (B), we observe that during the first 5 minutes, when the robot is subjected to predation, the cortisol level steadily rises and continues to increase even after the stimulus has ceased, including in the absence of any stimuli.

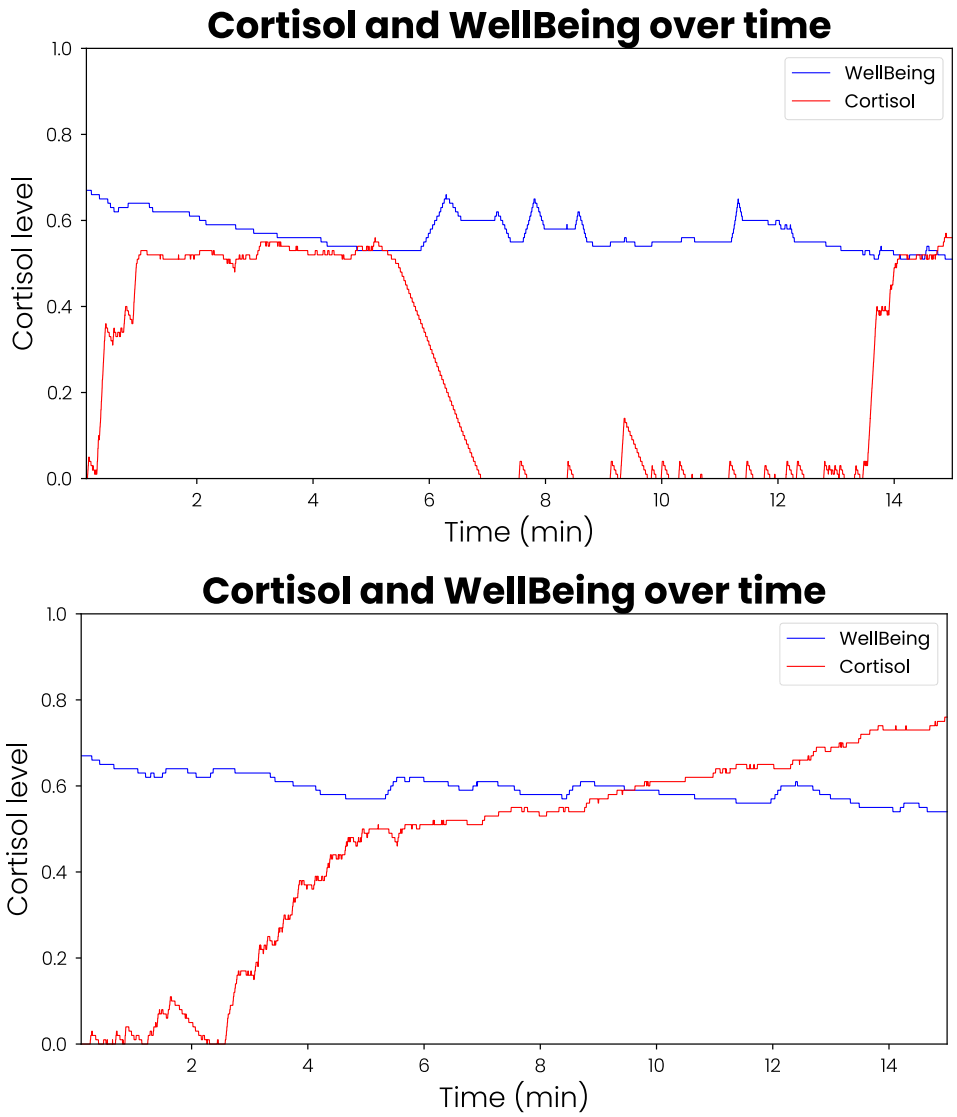


Figure 6.14: Intensity of Cortisol and Wellbeing over Time with Early Life Predation: No Cortex (top) and Cortex activity (bottom)

These results indicate that the presence of body representation memory supports the habituation and sensitization mechanisms we observed in Chapter 4. Before the sensory representations are fully formed, these memory mechanisms are strengthened, especially when the robot is exposed to a high level of stress-related danger. The sensory representation forms quickly and accompanies the rise in cortisol levels, further enhancing sensitization.

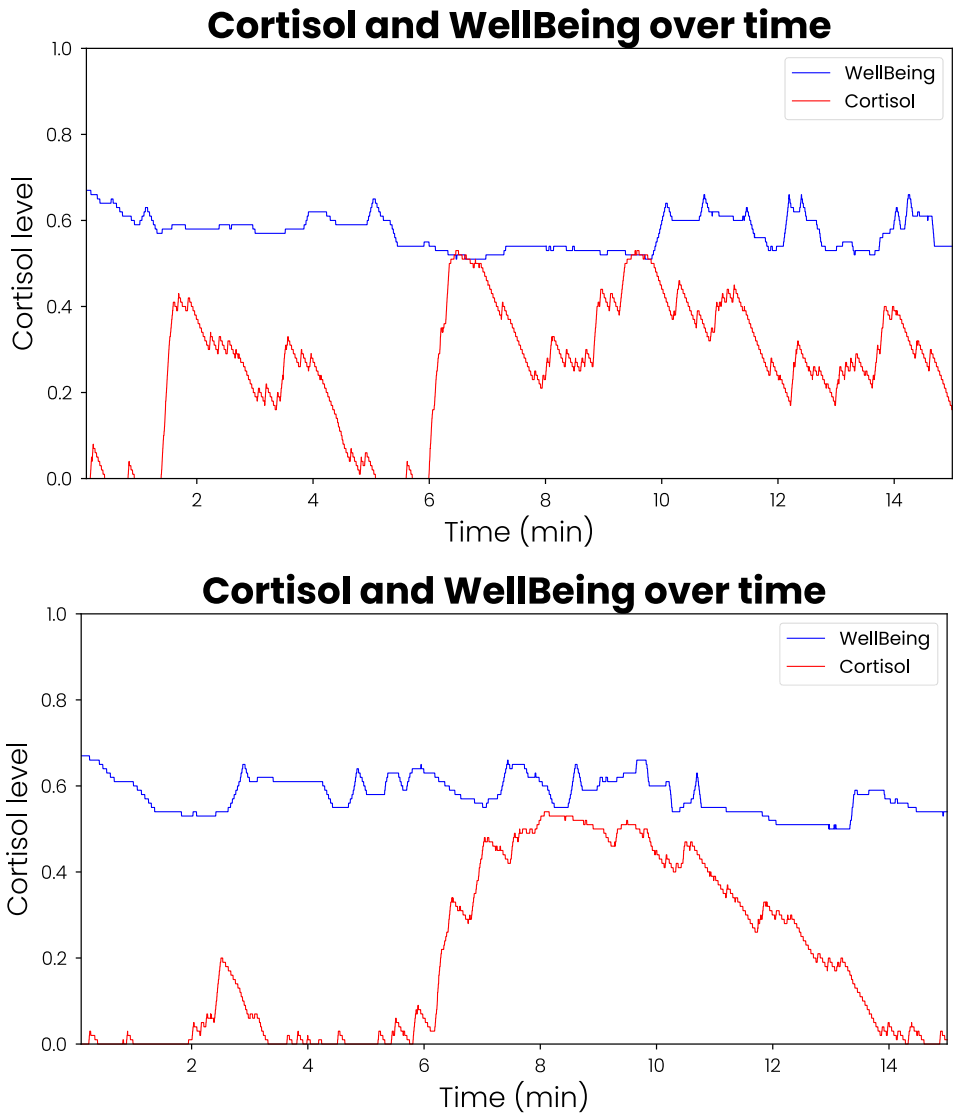


Figure 6.15: Intensity of Cortisol and Wellbeing over Time with Early Life Predation: No Cortex (top) and Cortex activity (bottom)

Though less intensely, we can also observe this phenomenon in a scenario where the stress-related danger is less severe, with two stalking predators, as shown in Fig. 6.15. The robot is not subjected to constant, intense stress in this scenario. In Fig. 6.15 (top), we observe that without the cortex, cortisol levels remain high but fluctuate over time, while with the cortex, cortisol levels stay elevated for a more extended period before gradually decreasing.

6.3.5 Activity Cycles in the physiological space

In Figs. 6.16 and 6.17, which represent the 3D Activity Cycles, we observe that in the scenario with 2 Hunting Predators lasting 15 minutes, the management of homeostatic variables was more or less satisfactory, both with and without the cortex. These new 3D A.C. (Activity Cycles) provide a reading of how the physiological spaces are managed

over time. When the points remain inside the pyramid that defines the stability cycles, the robot's homeostasis is maintained within a safe zone. When it exits, it indicates that one of the three physiological spaces is misaligned with the others, representing a danger, as one or more deficits are too high, potentially leading to the robot's death.

In these scenarios, the survival rate was 100% with and without the cortex, but it is clear that in the case without the cortex (Fig. 6.16), the deficits extend outside the stability zone. This is the mechanism by which the robot cannot maintain its internal homeostasis in environments with more predators and eventually dies. The cortex thus appears to be helpful in maintaining internal homeostasis.

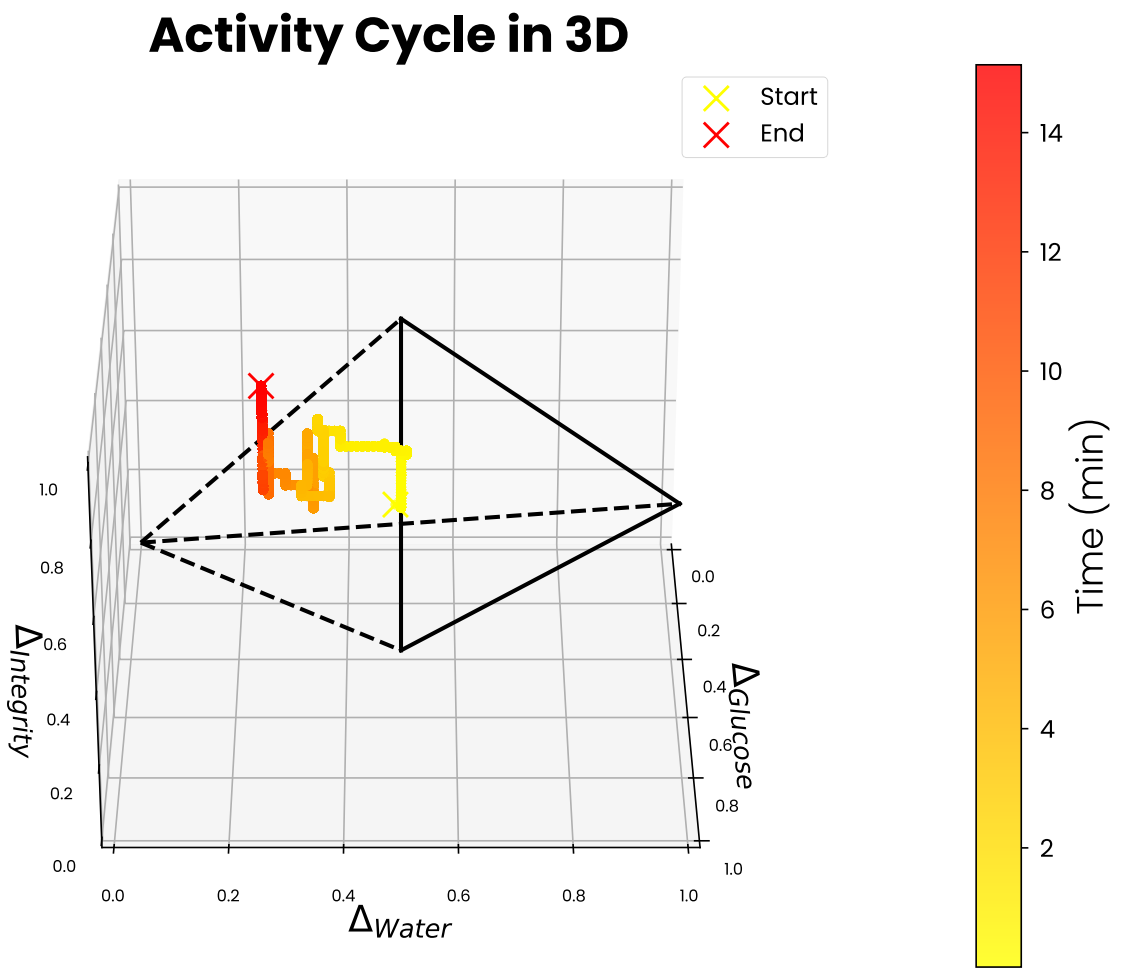


Figure 6.16: Activity cycles for the 15 minutes Hunting predator scenario without cortex, $\Delta_{glucose}$ is compared to Δ_{water} and $\Delta_{integrity}$ pyramid shape represents the stability cycles, going outside its area represents danger (with high deficits for one, two or all variables, respectively) in the physiological space

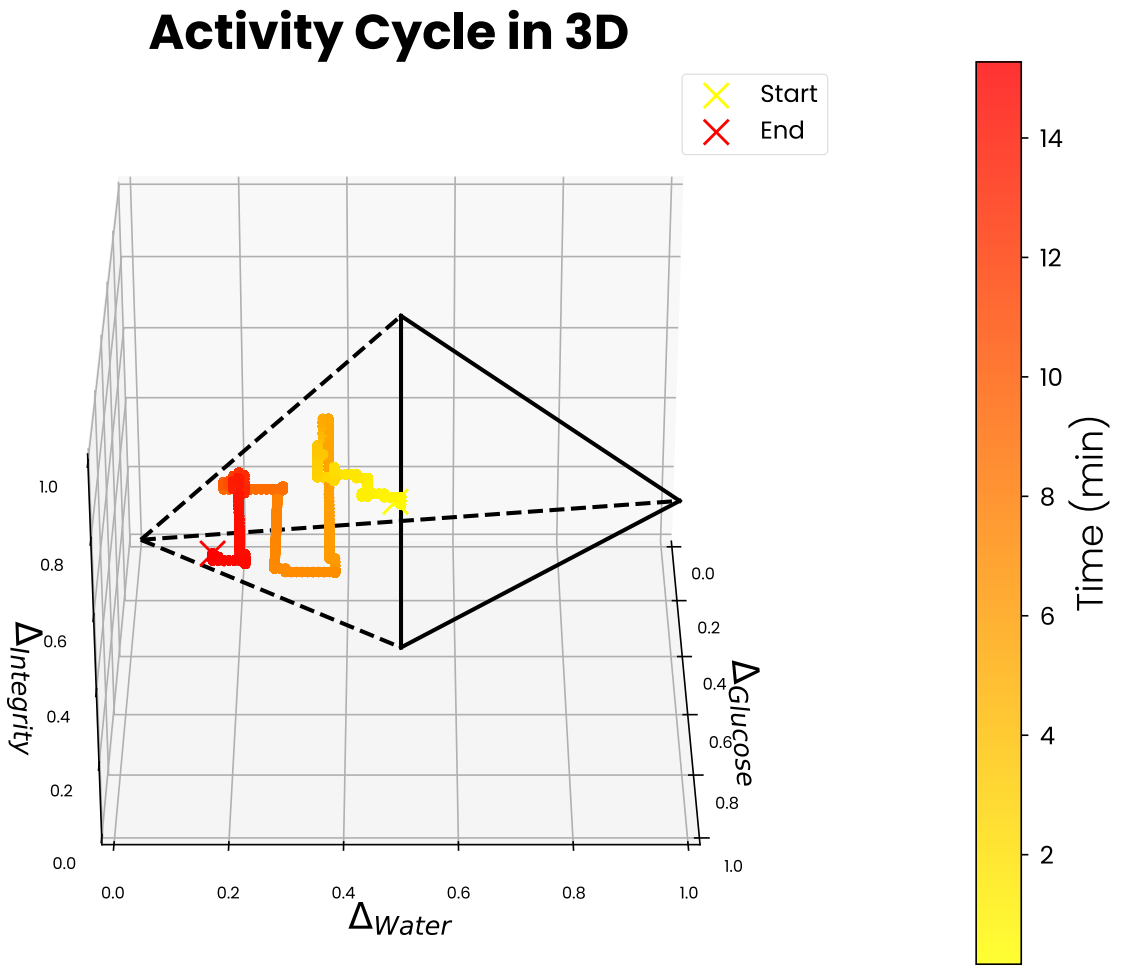


Figure 6.17: TheActivity cycles for the 15 minutes Hunting predator scenario with cortex, $\Delta_{glucose}$ is compared to Δ_{water} and $\Delta_{integrity}$ pyramid shape represents the stability cycles, going outside its area represents danger (with high deficits for one, two or all variables, respectively) in the physiological space

We can even observe in scenarios with the cortex examples of hyperstability of the cycle, where the robot manages to perfectly maintain its activity cycles within the permitted physiological space, sometimes reducing the deficits to a minimum. This is the case in Fig. 6.18, where in the 15-minute scenario with 2 Stalking Predators, the robot perfectly maintains its deficits within the viability space and continuously reduces the deficits until they are kept near zero.

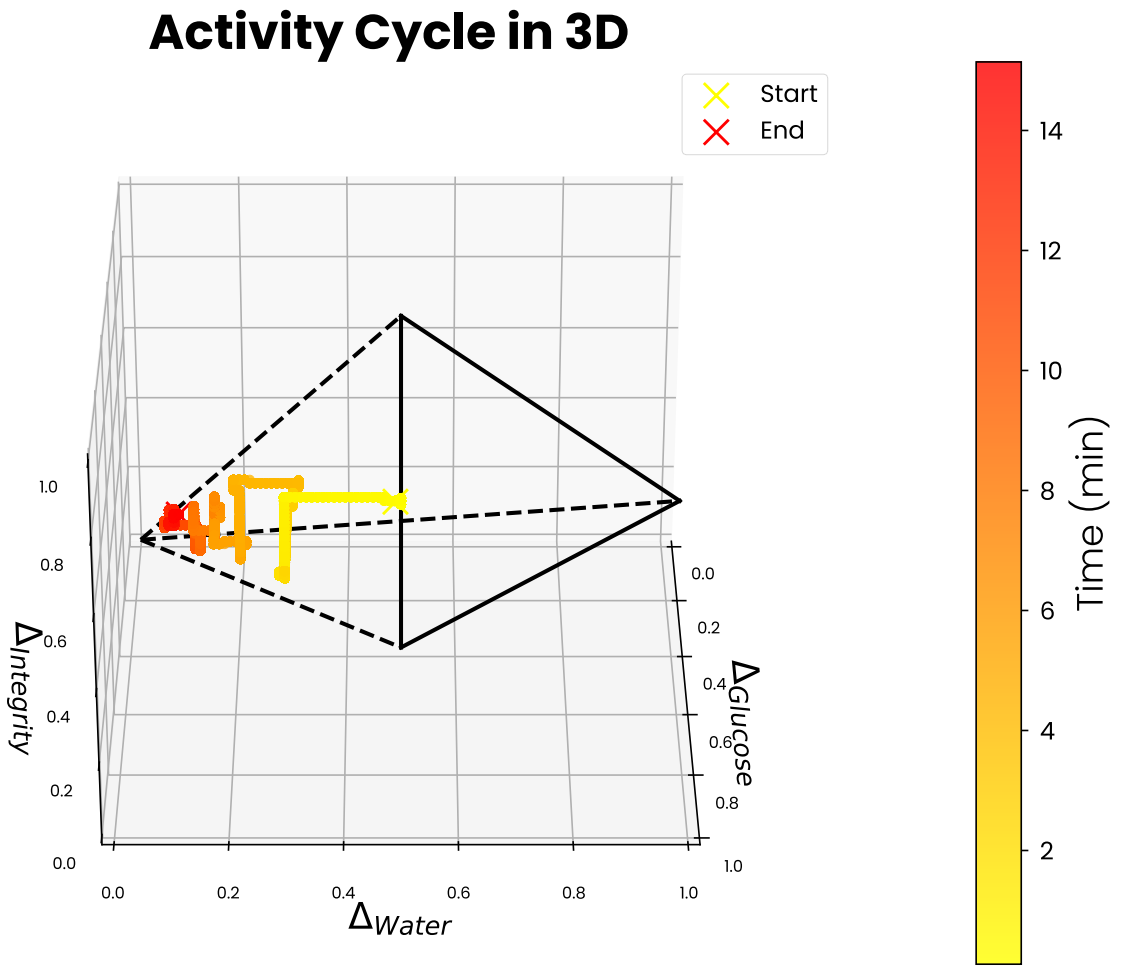


Figure 6.18: Activity cycles for the 15 minutes Stalking predator scenario with cortex, $\Delta_{glucose}$ is compared to Δ_{water} and $\Delta_{integrity}$ pyramid shape represents the stability cycles, going outside its area represents danger (with high deficits for one, two or all variables, respectively) in the physiological space

6.4 Discussion

In this chapter, I aimed to combine our previous work on the hormone-modulated action selection model with our work on constructing embodied internal representation. I also integrated the sensory field of nociception and mechanoreception, as described in the previous chapter, into our action selection model.

The results I presented emphasize the relevance of adding this bio-inspired cortex to our hormonal modulation layer within the framework of the homeostatic survival problem we addressed. In terms of survival rate and activity cycles, the model's overall performance has improved.

When observing cortisol levels, we can also see that the hormone enables finer and smoother regulation, which better considers the agent's history by providing a bodily representation and memory of it.

Finally, we observed that the concept of sensory representation, introduced in the previous chapter, makes sense in this social context of interaction with other robots. There is little interaction in a low social context (i.e., no predators), and the agent struggles to learn its sensory representations. In contrast, in rich social contexts with frequent interactions—whether from multiple predators or a single predator interacting repeatedly—we observe the segregation of features in the cortex and the emergence of complex sensory representations. These representations enable the robot to respond better to the survival problem posed.

Given that I have created systems for complex representation, we could explore the addition of a positive hormonal dimension, such as oxytocin, which could bring additional behavioral changes by responding to positive touches. This would introduce a new dimension to the social context we have studied.

From these sensory representations, we can also question the disorders that could arise in these representations. For example, if a sensor stops functioning, we should observe a phantom pain mechanism. Furthermore, we could investigate body perception disorders, such as dysmorphia, which could have a tangible impact on action selection. These research topics could be explored in future studies after the completion of this thesis.

Chapter 7

Conclusion

This thesis's core objective was to integrate the model of pain and pleasure into robotic architecture to explore how these perceptions can influence action selection, the process by which agents choose the appropriate behavior based on internal and external stimuli. This research had the twofold objective of using robots as models to contribute to the understanding and testing of psychological theories of pain and to develop more subtle decision-making strategies for autonomous and social robots.

In this thesis, I proposed to overview this topic with different perspectives on the theories of pain perception. I propose an action selection model miming pain perception's behavioral consequences; I investigate how such features can be adaptive or maladaptive in different environments. I studied how artificial hormones such as cortisol can modulate pain perception and introduce temporal elements to our models, leaning toward more adaptive action selection. Based on the current sensory limitation of robots, I proposed to build a sensory model for mechanoceptive and nociceptive processes to reconstruct complex information about tactile information. Finally, II proposed a cognitive architecture built on a spiking neural network to develop embodied construction of body representation with different areas of the brain.

Action selection is crucial to the survival of biological entities, such as humans or animals, and artificial agents, such as robots. Emotional experience, such as pain, is part of the bio-regulatory mechanisms involved in maintaining the stability of an organism's internal environment and its homeostasis [70, 131]. Building on motivation-based architecture [10], I proposed and developed around the different chapters an action selection architecture for a robotic agent that aims to evolve in a dynamic environment. It will face predation-related danger in this environment and must consume resources to survive. This basis framework introduced several bio-inspired mechanisms such as behavioral impact, artificial hormonal modulation, cognitive architecture, or other biological mechanisms. I discussed how these mechanisms have improved model performance or viability, how they influenced its success in maintaining internal homeostasis, and highlighted some emerging behaviors.

Pain is an unpleasant sensory and emotional experience linked to actual or potential damage [13]. It is a personal experience related to past and previous experiences and sociocultural perceptions. Its social impact can affect how we interact with each other [88]. Experiencing the same noxious stimuli one day and the other will not lead to the same

experience as each individual's internal state, memory, and context evolve. Proposing the pain perceptions model involved several challenges to mimic both behavioral responses and the underlying mechanisms responsible for pain perception.

Sensing the damage was one of these challenges, as in humans, tactile information is built on a diverse and wide range of sensory inputs and biological processes such as mechanoception and nociception. These processes allow the brain to detect where tactile stimuli are induced but also how they are induced, giving precious information about temperature, texture, pressure, sharpness, and chemical [4]. Robots' actual limitations do not offer such possibilities, so we needed to propose mechanisms that model these biological processes built on current robot sensory possibilities.

In this chapter, I will summarize briefly and discuss the key findings of our research. I will also discuss the limitations of our models and draw some perspectives.

7.1 Summary

In this thesis, I proposed to study how pain perception can affect action selection from different perspectives. To do so, I built on a prior motivation-based action selection architecture. However, I added several new elements to this architecture to modulate it with different aspects of pain. Pain is often neglected in affective models, even if it is crucial for survival. I proposed to compute pain perception not as a direct consequence of damage but as an emotional state modulated by various elements.

I proposed a novel model of **artificial nociceptors** (Chapter 3) that detected from the peripersonal sensorial space different types of noxious stimuli. I also proposed in this chapter a first computational model of the **pain emotional state** based on artificial hormone and how it can **impact** both action selection and **behavioral response** to noxious stimuli.

In Chapter 4, I proposed adding an **artificial simulated cortisol hormone** to our robotic model. I proposed to give this artificial hormone the role of stress regulator with a relationship to both artificial nociception and internal wellbeing. Adding **negative feedback** to its release, I proposed a computational model that leads to temporal dynamics such as habituation or sensitization.

In Chapter 4, I also proposed to compute **pain perception as the result of both the sensory process of noxious stimuli** with enhanced artificial nociceptors from Chapter 3 adapted to a new robotic platform, but also as a state **influenced by previous state with artificial cortisol acting as a direct modulator** of the perceived pain.

In Chapter 5, I proposed a new framework for the sensory perception of tactile and noxious stimuli in mobile robotic platforms. I proposed a **"sensory field"** from which I developed a new nociceptive and mechanoceptive process. This process allows us to **reconstruct complex information** about **where** and **how** tactile and noxious stimuli are induced to the robot.

In collaboration with Raphaël Bergoin, we proposed in this chapter a robotic architecture that was able to learn the embodied sensory representation with a neural network, leading

to the segregation of two areas representing different functions of the cortex linked to different stimuli.

In Chapter 6, I proposed to integrate the sensory field mechanoceptive, nociceptive sensory process, and neural cortex architecture I developed in Chapter 5 into our action selection model. I proposed to use the **neural network activity to compute pain perception**, keeping cortisol and wellbeing as modulators of perceived pain for robots. I also propose to integrate sensory field elements into our navigation algorithm. In this chapter, I also propose to use cortisol as a neuromodulator for physiological variables with gluconeogenesis. The final architecture I propose is graphically summarized in Fig. 7.1.

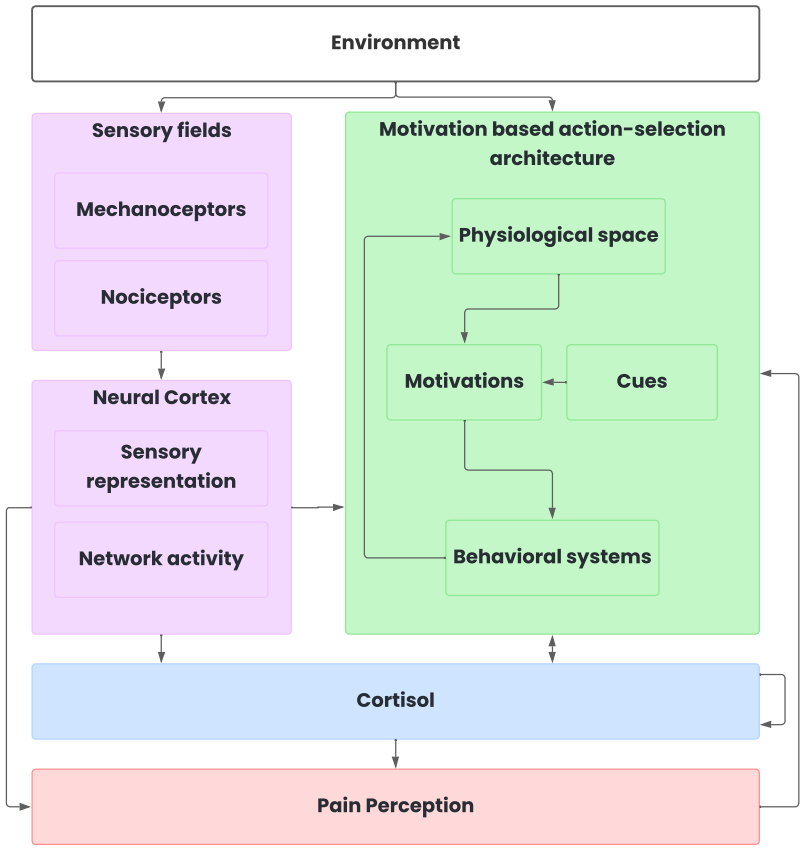


Figure 7.1: High-level diagram and graphical summary of our model and the effects I investigate in different chapters. This overview represents the final model presented in Chapter 6. We study Green elements in Chapter 3, Blue elements in Chapter 4, magenta elements in Chapter 5, and red elements are studied across all chapters.

In the next sections, I will furthermore develop and discuss how the contribution we propose can impact the action selection model and highlight some of the key findings we made. However, I synthetize here the key findings from our investigations that may provide helpful insides for embodied AI and affective computing robotics models:

- Pain perception is critical to enhance decision-making in dynamic and dangerous environments by providing robots a mechanism to balance survival-driving action or seeking resources (Chapter 3,4,6).

- Adaptive value of pain in environments with high levels of danger (e.g., predatory threats) and particularly hyper-sensitive pain perception in extreme environments (Chapter 3).
- Simulated cortisol hormonal modulation of pain and behavior led to emerging behaviors of fight-or-flight and laziness in extreme environments with respectively high and low levels of danger (Chapter 4).
- Hormonal memory with negative feedback can lead to habituation or sensitization mechanisms, helpful to adapt to a wide variability of environments (Chapter 4).
- The segregation of two different areas in a neural cortex linked to different sensory features (mechanoceptive and nociceptive processes) (Chapter 5).
- Earlier ("early life") experience of social interaction influences embodied sensory representation that can lead to different behavioral responses and enhance or reduce survival-related features (Chapter 6).

7.2 Adaptive value of pain

In Chapter 3, I proposed the first bio-inspired motivation-based action selection model for a robot that needed to evolve in a dynamic environment with different resources and predation-related danger. I introduced pain perception based on damage, building on the idea that pain was an artificial hormone whose release rate depended on different correlations between damage and pain experience. With this, I wanted to study how normal-correlated, hypo-correlated, or hypo-correlated perception of pain can influence survival and maintenance of the internal viability of our model. Damage was perceived with artificial nociceptors built on proximity sensors and sensitive to two types of noxious stimuli (impact and scratch).

Discussing Walter's and Williams's theory [88], which states that chronic pain may result from a mismatch between actual modern environments and pain systems, I tried to question the adaptive value of pain in different levels of pain-related danger environments.

I proposed different environments with different levels of danger, trying to mimic more ancestral environments with a lot of predation-related obstacles and more modern with abundant resources and no predation-related danger or obstacles. With obstacles, I introduced a dual impact of resource consumption, as the robot was able to perceive damage and pain; with the grooming behavior, if the robot were consuming too fast or compulsively the resource, it would experience pain.

Our results showed that in every environment, extreme correlations between damage and pain led to reduced variability of lifespan, highlighting that these perceptions led to behavioral patterns that can both improve or reduce the model's viability. We also saw that in modern environments with low levels of danger, the less the pain was perceived, the more the model adapted and survived longer. On the other hand, in ancestral environments with high levels of danger, the more the pain was correlated to damage, the greater the lifespan and the viability of the model. These results highlight how hyperacuity and pain sensitivity are remarkably adaptive in hazardous environments with high levels of danger.

Finally, we compared our model to the one initially built for humanoid robots by Maniscalo and Infantino [46] and highlighted its overall better performance regarding viability and correlation between damage and pain for the specific challenge we designed.

7.3 Hormonal neuromodulation

In Chapter 4, I proposed studying how artificial hormones influence pain perception. Inspired by Jean-Didier Vincent [44], I proposed studying cortisol, the stress hormone, as a new component of our model.

I modified the action selection architecture we built, adding this artificial hormone of cortisol that is sensible to damage predator-induced stress and to the general comfort level of the model (i.e., its internal management of homeostasis). This artificial hormone has a twofold impact on our model. First, it prioritizes resource-seeking behaviors, aiming to reduce stress linked to the discomfort of internal homeostasis, and second, it influences the salience of environmental cues based on the robot's current physiological state and needs, reinforcing the perception of predator danger and physiological resources.

I proposed that pain is no longer directly correlated to artificial nociceptors' induced damage but that cortisol is a modulator of pain perception. This made stress a factor in the robot's pain perception, reinforcing some of its behavioral impact.

I also introduced temporal elements, with previous cortisol concentration levels influencing the future release rate of cortisol; this led to habituation and sensitization mechanisms. We observed that in early life experience with low levels of stress-induced predation, cortisol levels remained low all along the experimental run, even with noxious stimuli encountered after, emphasizing a habituation mechanism. On the other hand, high levels of stress-related predation in early life led to sensitization with high levels of cortisol that do not lower, even when no more noxious stimuli are experienced.

We also studied the model's general performance, looking at Activity cycles, and highlighted how artificial cortisol helped the robot better maintain its internal homeostasis and minimize discomfort. In terms of survival rate, general performance also highlighted the adaptive value of neurohormonal modulation of pain perception with artificial cortisol, which improved survival.

We were able to observe two distinct emerging behaviors in specific experimental conditions. In high-threat environments, the agent developed a "fight" behavior, where, rather than avoiding predators, he occasionally made contact with the predators, repelling them. These behaviors appeared in scenarios where the agent had a high cortisol level and a perception of close resources and chose to consume resources over fleeing the predators, leading to this repulsive contact. This highlights how, in this situation, hormonal neuromodulation allowed robotic agents to develop a fight-or-flight strategy [79] in order to survive dynamic and challenging situations. We also observed emerging behaviors in low levels of stress-related danger environments where robotic agents maintained their internal homeostasis in comfort zones and began to linger around a resource instead of exploring the environment. This "laziness" behavior was only interrupted by the presence of predators. Extreme case environments led to diametrically opposed behaviors, emphasizing the interest of neurohormonal modulation of action selection.

7.4 Construction of embodied representations

In Chapter 5, we pointed out the limitations of current robot sensory information in reconstructing complex tactile information and noxious stimuli. We also pointed out the limits of the approach to creating sensors that mimic skin capacities, as they remain too experimental to be embodied in actual robots.

We proposed a new way to analyze tactile information in mobile robots with a sensory field body, representing a sensorial body around the robot from which we could reconstruct information about tactile signals. This idea uses the peripersonal space of the robot as a deformable skin. We built on proximity sensor information on two different sensing processes. Mechanoception, where we worked to analyse pressure, strain, and stress about the field to generate a vector of localized information about the tactile stimuli. Nociception, where we worked on physical information about deformation, frequency, velocity, and duration to build a vector that classifies the type of tactile stimuli the robot perceives.

As a joint work with Bergoin [124, 113] we proposed a spiking neural network that processed these sensory signals. We designed an experimental setup to stimulate the robot with every class of touch in every localization and observed how the network responded and learned.

We observed during the learning stage that we could identify the neurons associated with the different nociceptive features and the mechanoceptive localization. We were able to differentiate in time these signals as the spinal cord does it with different speeds of transmission between sensors and cortex [51]. Looking at the weighted connectivity, we observed that two different connectivity patterns formed, highlighting the segregation of two different areas of the cortex dedicated to specific tasks. This mimics the role of somatosensory cortex and anterior cingulate cortex. Looking more specifically at the somatosensory cortex, we could observe a circular pattern highlighting that the cortex has learned the physical representation of the robot, a circular robotic mobile platform. This bodily representation offers a different perspective from the solution proposed by Hoffman et al. [57] to represent somatosensory-like bodily representation constructions using a spiking neural network with temporal dynamics we could exploit.

7.5 Social context

Finally, in Chapter 6, I proposed a global action selection architecture that used the sensory field artificial nociception and mechanoception and cognitive architecture we developed in Chapter 5, the neuro-hormonal modulated action selection we discussed in Chapter 4. I aimed to use the construction of embodied representation to influence the social response of our robotic agent to social interaction, here, interaction with predators.

I highlighted how different social environments with different types of predators and different numbers of predators led to differences in the construction of embodied representations. In environments with low levels of social contact, robotic agents experienced minimal tactile contact. They could hardly learn their bodily representation, leading to a lower capacity to adapt to dynamic environments. On the other hand, environments

with high variability and much social interaction led to observing the segregation of the features in the cortex and the emergence of complex sensory representations. These different features helped the robotic agent maintain internal homeostasis by making it better respond to environment variability and interaction with the other social agents (the predators).

Our results emphasized the relevance of adding a neural cortex to our action selection model, as we observed improvements in our agent's survival rate in every type of social context and level of predation. Observing the activity cycles, we could observe that the learning of internal representations helped the robotic agent manage its internal homeostasis and minimize discomfort.

We also highlighted how the embodied representations interplay with artificial cortisol hormone, leading to finer and smoother regulation mechanisms, which took into account the history of robotic agents by learning the bodily representation. This particularly highlighted the role of early life experience in both learning and the capacity to adapt to dynamic environments.

7.6 Limitations & Perspectives

In this thesis, I have presented a multi-perspective study of how pain perception and its related mechanisms can improve motivation-based action selection architecture. I have focused on different mechanisms, such as artificial hormone modulation, artificial nociceptive sensing, behavioral impact, and cognitive architecture. I have proposed a reproducible and adaptive framework to study pain perception and its impact on robotic agents.

The model draws inspiration on ethology, clinician psychology, neurobiology, and computational neurosciences. Although I did not use this framework to study many clinician theories, I could use it as a basic framework to study chronic pain as we draw a first approach to the subject in Chapter 3.

I also could use the bodily representation we create to study dysfunctionalities of the pain perception system, such as the phantom pain [132]. Phantom pain is the experience of pain in a lost member due to an actual learned representation of the body and applies to our neural network model. I could also study dysmorphia [133] mechanisms, how different bodily representation perceptions can affect both pain perception and action selection mechanisms, and the complex interplay this can have in dynamic environments that could influence the dysmorphia itself.I

I could evaluate the frugality, regarding the computational cost and the energy efficiency, of our model and compare it to other models with different approach. I hope this work, and its multi-perspective approach to pain perception, can improve action selection at lower computational cost—offering low energy and low carbon mechanisms to improve actual decision-making systems that other searcher can build on.

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