

الجمهورية الجزائرية الديمقراطية الشعبية
Democratic and Popular Republic of Algeria
وزارة التعليم العالي والبحث العلمي
Ministry of Higher Education and Scientific Research
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Dissertation Presented for obtaining the MASTER diploma
In Biological Sciences
Specialty: Biochemistry

SLEEP DEPRIVATION AS A CONTRIBUTING FACTOR TO INSULIN RESISTANCE AND TYPE 2 DIABETES

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Sustained: June 2026

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Academic year: 2025-2026

Dedication

To all those who hold a special place in my heart, and to everyone who accompanied me along this journey and had a role in this success, I dedicate this humble work:

To Almighty Allah, Lord of the worlds, who blessed me with life and faith, and granted me strength, patience, and determination to complete this work. To Him belongs all praise and gratitude, now and forever.

To my dear father, a symbol of generosity and the source of my pride, who always saw in my simplest achievements the greatest successes, and who has always been my unwavering support. To you, I dedicate this work as a token of gratitude and appreciation for all your priceless love and sacrifices.

To my beloved mother, the source of tenderness and strength, who has always been the light that illuminates my path with her prayers and encouraging words. To you, who taught me patience and perseverance, I dedicate this achievement with all my love and respect.

To my dear sister Bouchra, the companion of my soul and a source of comfort, thank you for always being by my side and for your invaluable support throughout my journey.

To my dear brother Mohamed, who has always been a source of strength and support, I express my deepest gratitude. Thank you for your presence, encouragement, and unconditional support in both difficult and happy times.

To my friend and companion Sabrina, who was my refuge in moments of doubt and my support in times of weakness, thank you for your rare loyalty and invaluable support. I also extend my sincere gratitude to Moutassim for his companionship, support, and valuable advice, which were a great help throughout this journey.

To my dear friends Faiza and Maria, who shared with me every detail of this journey between joy and challenge, and whose presence was a source of strength and comfort that I will never forget.

To my dear doctor Salem Iman, who was like a mother to me, I extend my deepest gratitude for her moral support and continuous encouragement, which had a very positive impact on me.

To everyone I crossed paths with during this journey, whether close or far, I say: you have all contributed to reaching this moment and made the path smoother and more hopeful.

Finally, I dedicate this work to myself, in recognition of all the effort I have made, and in belief that this achievement is only the beginning of an endless journey of ambition.

Acknowledgements

First and foremost, I give thanks to Almighty Allah for granting me health, patience, and perseverance to successfully complete this work.

I would like to express my sincere gratitude to my supervisor, Professor **Kebir Nasr Eddine**, for the trust he placed in me, for his constant availability, as well as for his valuable guidance, insightful advice, and continuous support, which greatly contributed to the completion of this research.

I would also like to extend my deepest thanks to Dr. **Zaoui**, at the public hospital of **Sidi Boubekeur**, for his warm welcome, his supervision during my internship, as well as for his professional advice and support, which significantly enriched my practical experience.

My sincere thanks also go to all the staff of Sidi Boubekeur hospital for their cooperation, kindness, and team spirit, which made the internship period smoother and more productive.

I am also grateful to all my professors who contributed to my academic education throughout my university journey by providing knowledge, discipline, and scientific rigor.

Finally, I would like to express my heartfelt gratitude to my family and loved ones for their unwavering moral support, patience, and constant encouragement, which have been a true source of strength throughout this work.

Last but not least, I extend my thanks to everyone who, directly or indirectly, contributed to the completion of this work.

LIST OF ABBREVIATIONS

List of Abbreviations:

ADA: American Diabetes Association

BMI: Body Mass Index

CDC: Centers for Disease Control and Prevention

EEG: Electroencephalogram

ESS: Epworth Sleepiness Scale

GLUT4: Glucose Transporter Type 4

HbA1c: Glycated Haemoglobin

HOMA-IR: Homeostatic Model Assessment — Insulin Resistance

HPA: Hypothalamic-Pituitary-Adrenal Axis

IDF: International Diabetes Federation

IRS: Insulin Receptor Substrate

MODY: Maturity-Onset Diabetes of the Young

NIDDM : Non-Insulin-Dependent Diabetes Mellitus

NO: Nitric Oxide

NREM: Non-Rapid Eye Movement sleep

OGTT: Oral Glucose Tolerance Test

PI3K: Phosphatidylinositol 3-Kinase

PSQI: Pittsburgh Sleep Quality Index

RCT: Randomised Controlled Trial

REM: Rapid Eye Movement sleep

T2D: Type 2 Diabetes

WHO: World Health Organization

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ABSTRACT

ABSTRACT:

Background: Sleep deprivation is increasingly recognized as a modifiable risk factor for metabolic disorders. However, its relationship with insulin resistance and type 2 diabetes (T2D) remains insufficiently characterized, particularly among young adults and individuals with established diabetes.

Objectives: This study aimed to (1) assess sleep patterns and metabolic risk indicators among university health-science students and (2) evaluate the association between sleep quality and glycemic control in patients with type 2 diabetes.

Methods: A cross-sectional descriptive and analytical study was conducted among 196 participants, including 98 university students and 98 patients with T2D, recruited from academic and clinical settings in Saïda, Algeria. Data were collected using two structured questionnaires adapted from the Pittsburgh Sleep Quality Index (PSQI) and the Epworth Sleepiness Scale (ESS), together with sociodemographic, lifestyle, anthropometric, and clinical variables.

Results: Among students, 33% reported sleeping fewer than 6 hours per night, 72% experienced nocturnal awakenings, and a substantial proportion presented excess body weight. In the T2D group, 65% had HbA1c levels $\geq 8\%$, exceeding the American Diabetes Association target of $\leq 7\%$, indicating poor glycemic control. Furthermore, 52% of diabetic patients reported elevated blood glucose levels following poor sleep, while sleep fragmentation was highly prevalent in both groups. Overall, sleep disturbances were associated with several metabolic risk factors, including overweight status, reduced physical activity, and impaired glycemic control.

Conclusion: The findings highlight a significant association between sleep disturbances and metabolic dysregulation in both at-risk young adults and patients with type 2 diabetes. Sleep quality emerges as an important and potentially modifiable determinant of metabolic health and should be incorporated into preventive strategies and clinical management programs aimed at reducing insulin resistance and improving glycemic control.

Keywords: Sleep deprivation; Sleep quality; Insulin resistance; Type 2 diabetes; Glycemic control; HbA1c; Metabolic risk; University students; Sleep fragmentation; Saïda Algeria

ملخص:

الخلفية: يُعترف بشكل متزايد بحرمان النوم باعتباره عامل خطر قابلاً للتعديل يساهم في الاضطرابات الأيضية. ومع ذلك، لا تزال العلاقة بينه وبين مقاومة الإنسولين وداء السكري من النوع الثاني غير موصوفة بشكل كافٍ، خاصة لدى الشباب البالغين والأشخاص المصابين بالسكري.

الأهداف: هدفت هذه الدراسة إلى: (1) تقييم أنماط النوم ومؤشرات الخطر الأيضي لدى طلبة علوم الصحة في الجامعة، و(2) دراسة العلاقة بين جودة النوم والتحكم في سكر الدم لدى المرضى المصابين بداء السكري من النوع الثاني.

المنهجية: أُجريت دراسة مقطعية وصفية وتحليلية شملت 196 مشاركاً، منهم 98 طالباً جامعياً و98 مريضاً مصاباً بداء السكري من النوع الثاني، تم تجنيدهم من مؤسسات جامعية ومرافق صحية بمدينة سعيدة، الجزائر. جُمعت البيانات باستخدام استبيانين منظمين ومُكيّفين من مقياس بيتسبرغ لجودة النوم (PSQI) ومقياس إبورث للنعاس (ESS)، بالإضافة إلى متغيرات ديموغرافية واجتماعية، وعوامل مرتبطة بنمط الحياة، ومؤشرات أنثروبومترية وسريرية.

ABSTRACT

النتائج: أظهرت النتائج أن 33% من الطلبة ينامون أقل من 6 ساعات يوميًا، و72% يعانون من الاستيقاظ الليلي المتكرر، كما سُجلت نسبة معتبرة من زيادة الوزن لديهم. أما لدى المرضى المصابين بداء السكري من النوع الثاني، فقد أظهرت 65% منهم مستويات $HbA1c \geq 8\%$ ، وهي أعلى من الهدف الموصى به من قبل الجمعية الأمريكية للسكري ($> 7\%$)، مما يدل على ضعف التحكم في سكر الدم. إضافة إلى ذلك، أفاد 52% من المرضى بارتفاع مستويات الغلوكوز في الدم بعد ليلٍ من النوم السيئ، في حين كانت تجزئة النوم شائعة بشكل ملحوظ في كلا المجموعتين. وبصفة عامة، ارتبطت اضطرابات النوم بعدة عوامل خطر أيضية، من بينها زيادة الوزن، وقلة النشاط البدني، وضعف التحكم في سكر الدم.

الخلاصة: تُبرز هذه الدراسة وجود علاقة ذات دلالة بين اضطرابات النوم والاختلالات الأيضية لدى كل من الشباب المعرضين للخطر ومرضى داء السكري من النوع الثاني. وتُعد جودة النوم عاملاً مهماً وقابلاً للتعديل يؤثر في الصحة الأيضية، مما يستدعي إدماج تقييم النوم وتحسينه ضمن استراتيجيات الوقاية وبرامج الرعاية الصحية الهادفة إلى الحد من مقاومة الإنسولين وتحسين التحكم في سكر الدم.

الكلمات المفتاحية: حرمان النوم؛ جودة النوم؛ مقاومة الإنسولين؛ داء السكري من النوع الثاني؛ التحكم في سكر الدم؛ الهيموغلوبين السكري ($HbA1c$)؛ الخطر الأيضي؛ طلبة الجامعة؛ تجزئة النوم؛ سعيدة، الجزائر

RÉSUMÉ :

Contexte : La privation de sommeil est de plus en plus reconnue comme un facteur de risque modifiable des troubles métaboliques. Cependant, sa relation avec l'insulinorésistance et le diabète de type 2 (DT2) demeure insuffisamment caractérisée, en particulier chez les jeunes adultes et les personnes atteintes de diabète établi.

Objectifs : Cette étude visait à (1) évaluer les profils de sommeil et les indicateurs de risque métabolique chez des étudiants en sciences de la santé et (2) examiner l'association entre la qualité du sommeil et le contrôle glycémique chez des patients atteints de diabète de type 2.

Méthodes : Une étude transversale descriptive et analytique a été menée auprès de 196 participants, comprenant 98 étudiants universitaires et 98 patients atteints de DT2, recrutés dans des établissements universitaires et cliniques de Saïda, en Algérie. Les données ont été recueillies à l'aide de deux questionnaires structurés adaptés du **Pittsburgh Sleep Quality Index (PSQI)** et de l'**Epworth Sleepiness Scale (ESS)**, ainsi qu'à partir de variables sociodémographiques, liées au mode de vie, anthropométriques et cliniques.

Résultats : Parmi les étudiants, 33 % ont déclaré dormir moins de 6 heures par nuit, 72 % ont rapporté des réveils nocturnes fréquents et une proportion importante présentait un excès pondéral. Dans le groupe des patients diabétiques, 65 % présentaient une $HbA1c \geq 8\%$, dépassant l'objectif recommandé par l'**American Diabetes Association** ($\leq 7\%$), ce qui témoigne d'un mauvais contrôle glycémique. De plus, 52 % des patients diabétiques ont signalé une augmentation de leur glycémie après une mauvaise nuit de sommeil, tandis que la fragmentation du sommeil était fortement prévalente dans les deux groupes. Globalement, les troubles du sommeil étaient associés à plusieurs facteurs de risque métabolique, notamment le surpoids, la faible activité physique et l'altération du contrôle glycémique.

Conclusion : Les résultats mettent en évidence une association significative entre les troubles du sommeil et la dysrégulation métabolique chez les jeunes adultes à risque ainsi que chez les patients atteints de diabète de type 2. La qualité du sommeil apparaît comme un déterminant important et potentiellement modifiable de la santé métabolique. Elle devrait être intégrée aux

ABSTRACT

stratégies de prévention ainsi qu'aux programmes de prise en charge clinique visant à réduire l'insulinorésistance et à améliorer le contrôle glycémique.

Mots-clés : Privation de sommeil ; Qualité du sommeil ; Insulinorésistance ; Diabète de type2 ; Contrôle glycémique ; HbA1c ; Risque métabolique ; Étudiants universitaires ; Fragmentation du sommeil ; Saïda Algérie.

GENERAL INTRODUCTION

Introduction:

Sleep is a fundamental biological function, essential for physical and mental health. It plays a key role in regulating many physiological processes, such as hormonal balance, nervous system function, energy metabolism, and the immune system (Medic, Wille & Hemels, 2017). Adequate, good-quality sleep also improves cognitive abilities, including memory, attention, and concentration (Diekelmann & Born, 2010). Thus, getting enough sleep is essential for maintaining the body's balance and ensuring its proper functioning.

During sleep, several essential mechanisms are activated to allow the body to recover and maintain its internal balance. Deep sleep, also known as slow-wave sleep, is the most restorative phase (Borbély & Achermann, 2000). During this period, heart rate and blood pressure decrease, as does sympathetic nervous system activity, while the brain consumes less glucose. At the same time, the secretion of anabolic hormones, especially growth hormone, increases, while cortisol decreases (Diekelmann & Born, 2010). All these mechanisms contribute to maintaining metabolic and energy balance.

However, modern lifestyles have gradually reduced sleep time and promoted late bedtime habits. Excessive use of electronic devices, academic and professional demands, and irregular daily rhythms all reduce sleep duration and quality. Several studies show that a large number of adults sleep fewer than seven hours per night, which is insufficient for optimal body function (Watson et al., 2015; CDC, 2016). In parallel, obesity and type 2 diabetes are continuously increasing worldwide, representing a major public health problem (Knutson & Van Cauter, 2008).

Numerous studies suggest that sleep deprivation is an important risk factor in the development of metabolic disorders (Spiegel et al., 2004). These disorders manifest as abnormalities such as dyslipidemia and hyperglycemia, thereby increasing the risk of type 2 diabetes (Reaven, 1988).

Sleep deprivation also disrupts the hormonal balance involved in appetite regulation, particularly leptin and ghrelin (Taheri et al., 2004). A decrease in leptin combined with an increase in ghrelin leads to greater hunger, potentially causing increased food intake and weight gain. Moreover, sleep deprivation impairs glucose metabolism and reduces insulin sensitivity, thereby promoting the onset of insulin resistance, a key mechanism in type 2 diabetes (Spiegel et al., 2004).

Thus, chronic sleep deprivation appears as a modifiable factor contributing to the development of metabolic syndrome, obesity, and type 2 diabetes. In this context, it is pertinent to ask to what extent sleep deprivation influences insulin sensitivity and the risk of developing type 2 diabetes.

This study aims to analyze the impact of sleep deprivation on the metabolic mechanisms involved in glucose regulation, as well as to evaluate its role in the development of type 2 diabetes.

RESEARCH PROBLEM

Research problem:

Modern lifestyles have progressively eroded sleep duration and quality across all age groups. University students face academic pressure, irregular schedules, and excessive screen exposure, while patients with chronic diseases such as type 2 diabetes suffer from condition-specific sleep disturbances like nocturia, neuropathic pain, and nocturnal hypoglycaemia. At the same time, the global prevalence of type 2 diabetes continues to rise at an alarming rate, representing a major public health burden in both developed and developing countries, including Algeria.

Despite growing scientific evidence linking sleep deprivation to metabolic dysfunction, sleep remains largely absent from routine clinical assessments and diabetes prevention strategies. The biological mechanisms are well established: insufficient sleep activates the hypothalamic-pituitary-adrenal (HPA) axis, elevates cortisol, disrupts leptin and ghrelin balance, and impairs GLUT4-mediated glucose uptake in skeletal muscle — all of which directly promote insulin resistance and glycaemic dysregulation.

Yet in the Algerian context, no study has simultaneously examined sleep profiles in an at-risk student population and assessed the sleep-glycaemia relationship in diagnosed diabetic patients using a comparative approach. This gap constitutes the core research problem: **the role of sleep deprivation as a modifiable, underrecognised contributing factor to insulin resistance and type 2 diabetes remains insufficiently documented in specific Algerian populations.**

Research questions:

General Research Question:

To what extent does sleep deprivation contribute to the development or worsening of insulin resistance and type 2 diabetes?

Specific Research Questions :

1. What are the sleep duration and quality profiles of university health-science students, and do these profiles reflect patterns associated with increased metabolic risk?
2. Is there an association between sleep duration of fewer than seven hours per night and indicators of glycaemic imbalance in university students?
3. In patients with established type 2 diabetes, is poor sleep quality associated with less optimal glycaemic control, as reflected by HbA1c levels?
4. What are the main causes of nighttime awakenings in diabetic patients, and how do these relate to their level of glycaemic control?

HYPOTHESIS

General Hypothesis:

Sleep deprivation may disrupt metabolic balance and contribute to the development or worsening of type 2 diabetes.

Specific Hypotheses :

1. A sleep duration of fewer than seven hours per night may be associated with an increased risk of glycemic imbalance.
2. Poor sleep quality may lead to hormonal disturbances, notably an increase in cortisol and alterations in growth hormone secretion.
3. Irregular sleep habits, such as late bedtimes and reduced sleep duration, may be common among students and may influence their metabolic balance.
4. In patients with type 2 diabetes, sleep deprivation may be associated with less optimal glycemic control.

HYPOTHESIS

Conceptual Definitions:

Diabetes mellitus is a chronic metabolic disorder characterized by persistent **hyperglycaemia** due to impaired insulin secretion, insulin action, or both.

Insulin is the key hormone that lowers blood glucose by promoting its uptake into cells.

Insulin resistance is a reduced cellular response to insulin and represents the central mechanism of type 2 diabetes.

β cells (pancreatic cells) produce insulin; their progressive dysfunction leads to inadequate insulin supply.

Blood glucose (glycaemia) reflects circulating glucose levels; fasting values ≥ 1.26 g/L confirm diabetes.

HbA1c indicates average blood glucose over the previous 2–3 months and is used to evaluate **glycaemic control**, which is essential to prevent complications.

Obesity is a major risk factor for insulin resistance. Adipose tissue releases **adipokines** (e.g., leptin, adiponectin) that regulate metabolism and inflammation.

Sleep and circadian rhythm play a critical role in metabolic regulation. The **circadian rhythm** controls the 24-hour cycle of sleep, hormones, and metabolism. Sleep deprivation or circadian disruption impairs glucose regulation.

Cortisol, a stress hormone, increases blood glucose and reduces insulin sensitivity; its levels rise with poor sleep.

Melatonin regulates sleep onset and circadian timing and is suppressed by light exposure at night.

Leptin (satiety hormone) and **ghrelin** (hunger hormone) regulate appetite. Sleep deprivation decreases leptin and increases ghrelin, promoting overeating and weight gain.

Acute imbalances include **hyperglycaemia** (high blood glucose) and **hypoglycaemia** (low blood glucose).

Chronic hyperglycaemia leads to major complications:

HYPOTHESIS

- **Neuropathy** (nerve damage)
- **Nephropathy** (kidney damage)
- **Retinopathy** (eye damage)

Sleep disturbances in diabetic patients are commonly caused by **nocturia** (nighttime urination) and neuropathic pain, further worsening metabolic control.

PART I: LITERATURE REVIEW

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I.1.1 Definition of Sleep:

Sleep is one of the fundamental biological functions essential for maintaining the physical and psychological balance of the human being. It is not merely a state of rest or inactivity, but a complex physiological process during which the body and brain undergo several important changes that contribute to energy restoration and the regulation of many vital functions. Numerous scientific studies have demonstrated that sleep plays an essential role in preserving health and quality of life. It is notably involved in regulating the nervous system, strengthening the immune system, maintaining hormonal balance, and improving cognitive abilities such as memory, concentration, and learning (Walker, 2017; Van Cauter & Spiegel, 1999).

Sleep is regulated by a set of complex biological mechanisms, among which the biological clock and circadian rhythm control the alternation between waking and sleeping periods throughout the day. This rhythm is influenced by several internal factors, including the secretion of hormones such as melatonin, as well as external factors such as light, lifestyle, and daily habits. Furthermore, sleep is composed of different successive phases, including slow or deep sleep, which promotes physical recovery and cellular regeneration, and REM sleep, characterized by rapid eye movements and playing an important role in memory consolidation and the regulation of cognitive and emotional functions (Carskadon & Dement, 2011).

In recent decades, changes associated with modern lifestyles have contributed to an increase in sleep disorders and sleep deprivation across many populations worldwide. Technological advancements, increasing professional and academic demands, as well as social pressures have altered the daily habits of many individuals, making sleep deprivation increasingly common. Excessive use of electronic devices, night shifts, and psychological stress are also factors that can impair sleep duration and quality (National Sleep Foundation, 2020).

Many studies have shown that sleep deprivation or disturbances can have harmful effects on physical and mental health. In the short term, sleep deprivation can cause significant fatigue, decreased concentration and attention, mood disturbances, and reduced cognitive performance. In the long term, it may be associated with an increased risk of developing several chronic diseases such as obesity, hypertension, and cardiovascular diseases, as well as certain metabolic disorders (Spiegel et al., 2005).

In this context, sleep duration and quality play an essential role in regulating the body's metabolic processes. Indeed, during sleep, several hormonal changes occur that influence energy balance and appetite regulation, particularly through the hormones leptin and ghrelin, which control hunger and satiety. Good-quality sleep also contributes to improving cellular insulin sensitivity, the hormone responsible for regulating blood glucose levels. Consequently, sleep disturbances or reduction can disrupt these physiological mechanisms, thereby increasing the risk of developing insulin resistance and type 2 diabetes (Tasali & Van Cauter, 2006).

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Given the importance of sleep and its influence on the various bodily functions, the study of sleep and its disorders has become a major field of scientific and medical research. Many studies today seek to better understand the mechanisms of sleep regulation, the factors that influence it, and its role in the development of certain chronic diseases, particularly metabolic disorders such as diabetes.

I.1.2 Sleep Phases:

Sleep consists of cycles that succeed one another throughout the entire night. A cycle lasts approximately 60 to 120 minutes and is renewed 3 to 6 times during the night in a 'good' night of sleep. These cycles are also divided into different phases such as waking, wake-sleep transition, REM sleep, and light or deep NREM sleep (Inserm, 2017). Each of these phases corresponds to a different brain activity revealed by electroencephalography. The two main ones are slow-wave sleep (NREM, non-rapid eye movement) and REM (Rapid Eye Movement) sleep.

I.1.2.1 NREM Sleep

Non-REM (NREM) sleep constitutes the initial phase of each sleep cycle. It is characterized by an absence or slowness of eye movements and is accompanied by a progressive decrease in brain activity and physiological functions, such as reduced heart rate, decreased blood pressure, slowed breathing, and diminished energy consumption. This reflects the body entering a state of deep relaxation. This type of sleep is subdivided into three stages (N1, N2, and N3), which differ in sleep depth and the intensity of associated physiological changes.

I.1.2.1.1 NREM Phases

Phase 1 (N1)

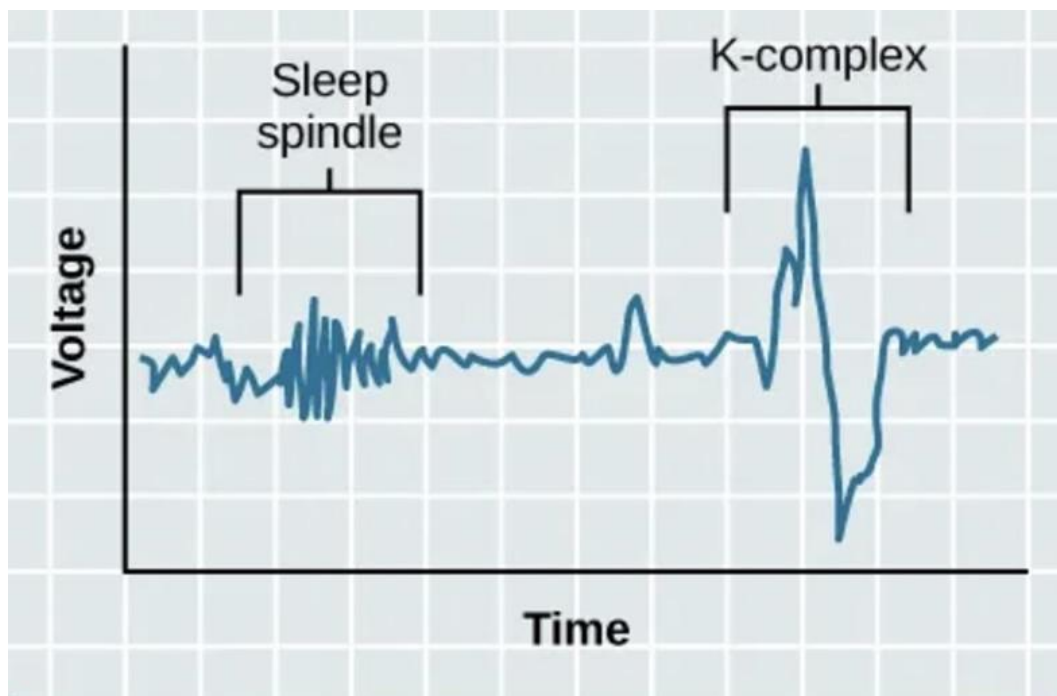
The N1 phase is a transition phase between wakefulness and sleep, characterized by a progressive decrease in neuronal activity and a slowing of eye movements. The brain is in a state of light relaxation, and this phase typically lasts between 1 and 7 minutes, representing approximately 5% of total sleep duration. Sleep is very light, allowing easy awakening. Among the characteristics of this phase, one sometimes notices a sudden falling sensation (hypnic jerk), resulting from muscle contraction, accompanied by the appearance of alpha waves on the electroencephalogram (EEG). These waves, with a frequency of 8–13 Hz, typically appear in the posterior region of the brain and indicate a calm state and the beginning of the transition to sleep.

Phase 2 (N2)

The N2 phase is the stage where the body enters more stable sleep, typically lasting 10 to 25 minutes and representing approximately 55% of total sleep. It is characterized by a decrease in body temperature, cessation of eye movements, and greater muscular relaxation. The brain exhibits specific activity during this phase, including theta waves (4–7 Hz), sleep spindles (short rapid waves of 11–16 Hz lasting approximately 0.5 to 2 seconds, arising from the interaction between the cortex and thalamus), and K-complexes (one of the most important electrical patterns in the brain during sleep, characterized by a rapid negative phase followed by a slow

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positive phase, lasting approximately 0.5 to 1 second). The primary function of K-complexes is sleep protection: the brain evaluates external stimuli, and if the stimulus is weak, the K-complex appears and sleep continues, avoiding unnecessary waking.



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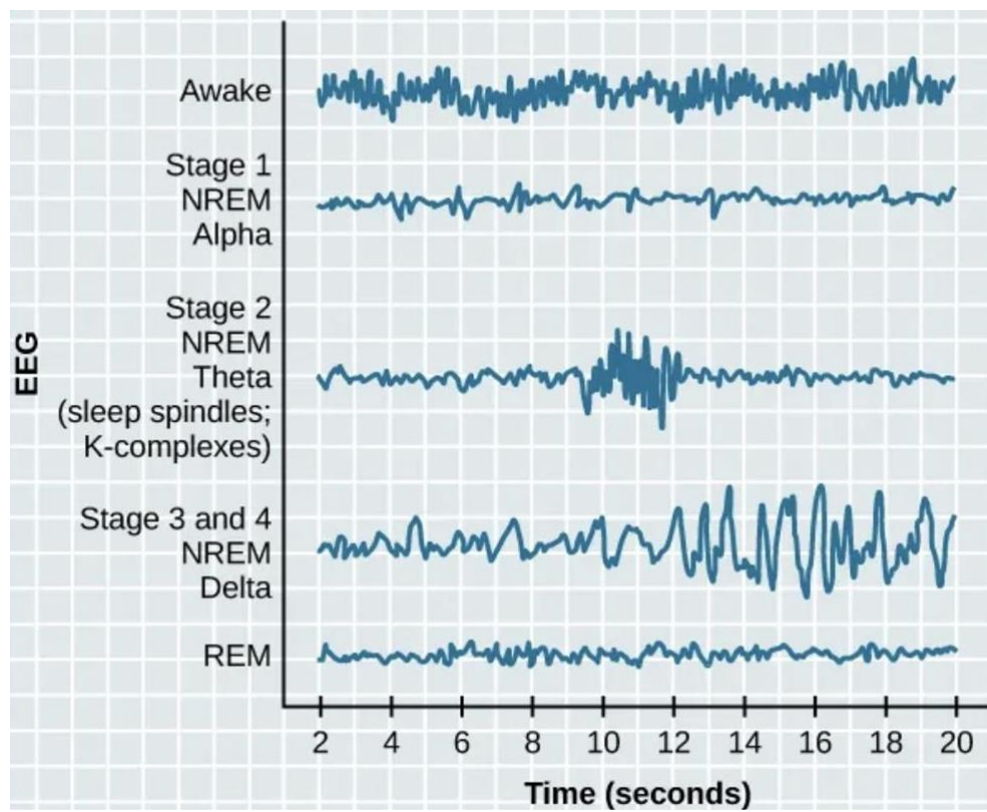
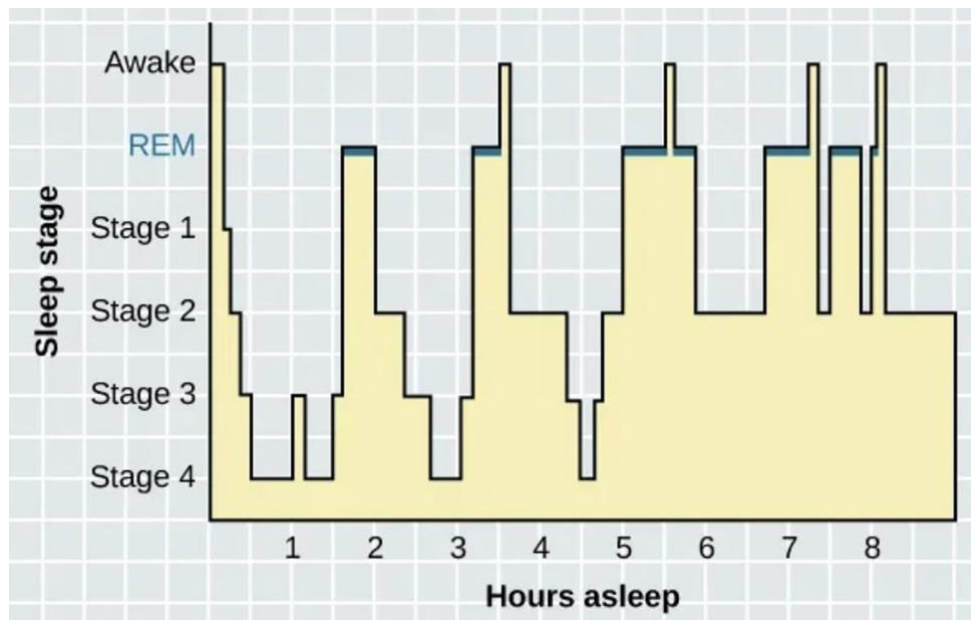
Phase 3 (N3) – Slow Wave Sleep

In the N3 phase, which represents the deepest stage of sleep known as Slow Wave Sleep, significant changes in brain and physiological activity are observed. Brain activity is characterized by the appearance of delta waves (0.5–4 Hz) of low frequency and high amplitude, reflecting a high degree of synchronization of cortical neurons. During this phase, neurons alternate between two successive states called Down state (marked decrease in neuronal activity) and Up state (synchronized and intense resumption of neuronal activity). Physiologically, this phase is accompanied by decreased heart rate, blood pressure, and breathing rate, almost complete muscle relaxation, and reduced energy consumption. It plays an essential role in energy recovery, growth hormone secretion, tissue repair, and memory consolidation, particularly declarative memory.

I.1.2.2 REM Sleep:

REM (Rapid Eye Movement) sleep is a phase of sleep characterized by rapid eye movements, intense brain activity close to the waking state, and near-complete muscular atonia that prevents the physical execution of dreams. This phase is therefore physiologically paradoxical, as it combines high brain activity with muscular immobility (Kryger, Roth & Dement, 2017).

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REM sleep plays an essential role in several cognitive and psychological functions. It participates in memory consolidation, particularly emotional and procedural memory. It is also involved in emotional regulation, helping to process and stabilize experiences from the day. Finally, it contributes to the processing and organization of acquired information, thereby promoting cognitive and behavioral adaptation (Diekelmann & Born, 2010; Walker, 2009).

I.1.3 Sleep Regulation:

I.1.3.1 Circadian Regulation

Sleep is regulated by an internal circadian system that acts as a biological clock. This system is controlled by the suprachiasmatic nucleus located in the hypothalamus, which synchronizes wake-sleep rhythms over a period of approximately 24 hours. It is strongly influenced by light: light exposure inhibits melatonin secretion, which promotes wakefulness,

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while darkness stimulates its production by the pineal gland, thus facilitating sleep onset and maintenance (Saper, Scammell & Lu, 2005; Kryger, Roth & Dement, 2017).

I.1.3.2 Hormonal Regulation

During sleep, several hormones participate in restoring the body. Growth hormone secretion increases during the night, particularly during deep sleep, which promotes tissue repair and growth processes (Guyton & Hall, 2016). Moreover, cortisol levels decrease at the beginning of sleep, thereby reducing alertness and physiological stress (Medic, Wille & Hemels, 2017).

I.1.3.3 Nervous System Regulation

The autonomic nervous system also plays an essential role in regulating sleep. During sleep, the parasympathetic system becomes dominant, leading to a decrease in heart rate, blood pressure, and alertness level. This physiological state promotes relaxation and body recovery (Kryger, Roth & Dement, 2017).

I.1.4 Factors Influencing Sleep:

I.1.4.1 Environmental Factors

Several environmental factors can impair sleep quality, including light and noise. Excessive exposure to light, especially in the evening, disrupts melatonin secretion and delays sleep onset. Similarly, nighttime noise fragments sleep and reduces its continuity, thereby diminishing its overall quality (Kryger, Roth & Dement, 2017).

I.1.4.2 Behavioral Factors

Lifestyle habits play an important role in sleep regulation. Screen use (phone, tablet) before bedtime exposes individuals to blue light, which inhibits melatonin production. Additionally, caffeine consumption stimulates the central nervous system and increases alertness, thereby delaying sleep onset and reducing sleep duration (Medic, Wille & Hemels, 2017).

I.1.4.3 Stress and Anxiety

Psychological stress and anxiety are major factors disrupting sleep. They increase sympathetic nervous system activation and maintain a state of hypervigilance, making it difficult to fall asleep and promoting nighttime awakenings (Saper, Scammell & Lu, 2005).

I.1.4.4 Irregular Schedules and Night Work

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Irregular sleep schedules and night work strongly disrupt the circadian rhythm. These desynchronizations between the internal biological clock and social schedules lead to reduced sleep quality, chronic fatigue, and impaired alertness (Kryger, Roth & Dement, 2017).

I.1.5 Sleep Disorders :

I.1.5.1 Insomnia

Insomnia is one of the most common sleep disorders. It is characterized by difficulty falling asleep, frequent nighttime awakenings, or early morning awakening with inability to go back to sleep. This disorder leads to a reduction in sleep duration and quality, which can affect cognitive performance and quality of life (Medic, Wille & Hemels, 2017).

I.1.5.2 Sleep Apnea

Sleep apnea is a disorder characterized by repeated interruptions in breathing during sleep. These respiratory pauses lead to frequent micro-awakenings, fragmenting sleep and reducing its restorative effectiveness. It is often associated with excessive daytime sleepiness and cardiovascular risks (Kryger, Roth & Dement, 2017).

I.1.5.3 Circadian Rhythm Disorders

Circadian rhythm disorders correspond to a desynchronization between the internal biological clock and the external environment. They can be caused by night work, travel with jet lag, or irregular schedules. This desynchronization leads to difficulties falling asleep, chronic fatigue, and impaired alertness (Saper, Scammell & Lu, 2005).

I.1.6 Importance of Sleep in Biological Balance:

I.1.6.1 Metabolic Regulation

Sleep plays a fundamental role in regulating metabolism. It participates in maintaining energy balance and influences several physiological processes, including glucose control and lipid metabolism. Insufficient sleep can disrupt these mechanisms and increase the risk of metabolic disorders (Medic, Wille & Hemels, 2017).

I.1.6.2 Immune Function

Sleep strongly contributes to proper immune system functioning. During sleep, the body promotes the production and activity of certain immune cells, improving defense against infections and strengthening the overall immune response (Kryger, Roth & Dement, 2017).

I.1.6.3 Energy Restoration

Sleep enables energy restoration of the body. It reduces overall energy expenditure and promotes physical and mental recovery. This phase is essential to restore physiological functions and maintain an optimal level of performance during the day (Guyton & Hall, 2016).

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I.1.6.4 Memory Consolidation

Sleep plays a key role in cognitive functions, particularly memory consolidation. During sleep, information acquired during the day is transferred and stabilized in the brain, thereby improving learning and long-term memory (Diekelmann & Born, 2010).

I.1.6.5 Glucose Regulation and Insulin Sensitivity

Sleep also influences glucose metabolism and insulin sensitivity. Poor quality or insufficient sleep can reduce insulin sensitivity and disrupt glycemic regulation, thereby increasing the risk of metabolic disorders such as type 2 diabetes (Spiegel et al., 2005).

I.1.7 Effects of Sleep Deprivation:

I.1.7.1 Short-Term Effects

Sleep deprivation causes several immediate effects on the body and cognitive functions. It leads to significant fatigue, decreased concentration, and mood disturbances, which can impair daily performance and alertness (Medic, Wille & Hemels, 2017).

I.1.7.2 Long-Term Effects

In the long term, chronic sleep deprivation is associated with several metabolic and cardiovascular pathologies, notably obesity, hypertension, and type 2 diabetes. These effects result from prolonged disruption of hormonal and metabolic mechanisms (Kryger, Roth & Dement, 2017).

I.1.7.3 Hormonal and Metabolic Imbalances

Sleep deprivation also leads to significant hormonal changes, characterized by a decrease in leptin (satiety hormone) and an increase in ghrelin (hunger hormone). This imbalance promotes increased appetite and weight gain. Furthermore, a reduction in insulin sensitivity may appear, increasing the risk of glycemic disorders (Spiegel et al., 2005).

I.1.8 Chapter summary

So, Sleep is an essential biological process for the proper functioning of the body. It contributes to maintaining physical, psychological, and metabolic balance. It occurs in cycles and different phases, each playing a specific role in recovery and proper body function. Several biological, hormonal, and nervous mechanisms interact to ensure its regulation. However, various environmental and behavioral factors can impair its quality and duration. Sleep disorders and sleep deprivation represent an important health problem, potentially leading to significant

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short and long-term consequences. Current research also shows that sleep is involved in metabolic regulation, particularly regarding insulin sensitivity and the risk of type 2 diabetes, a point that will be developed in the following chapter.

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Diabetes is a chronic condition of multifactorial etiology, clinically characterized by persistent hyperglycemia observed under normal dietary conditions, in the absence of intercurrent pathologies or treatments likely to cause a transient rise in blood glucose. Due to its high prevalence and the severity of its complications, this disease represents a major public health problem. Furthermore, its frequency continues to increase at an alarming rate worldwide (Blickle, 2014). According to the International Diabetes Federation (IDF, 2014), approximately 387 million people worldwide were affected by diabetes, of whom 46.3% remain undiagnosed.

In this context, the objective of our work is to study the impact of sleep deprivation as a factor contributing to insulin resistance and type 2 diabetes. More specifically, we analyze the distribution of subjects by sex, age, and body mass index (BMI), as well as by the presence of family history and associated complications.

We also compare various biochemical parameters, including blood glucose and HbA1c, between individuals with sleep disorders and those with normal-quality sleep, in order to evaluate the impact of sleep deprivation on carbohydrate and lipid metabolism.

Finally, we seek to highlight a possible correlation between sleep duration and quality, on the one hand, and HbA1c values as well as fasting blood glucose levels, on the other hand, in patients with type 2 diabetes.

I.2.1 Type 2 Diabetes and Metabolic Disorders:

Type 2 diabetes is one of the most common metabolic disorders and constitutes a major public health challenge worldwide. It is associated with alterations in carbohydrate and lipid metabolism and may lead to severe long-term complications if not adequately controlled

I.2.2 Definition and Diagnosis of Diabetes Mellitus:

Diabetes mellitus is defined by the World Health Organization (WHO) and the American Diabetes Association (ADA) as a group of metabolic diseases characterized by chronic hyperglycemia resulting from either a defect in insulin secretion, an alteration of its action, or both abnormalities combined. Chronic hyperglycemia is responsible, in the long term, for complications affecting primarily the eyes, kidneys, nerves, heart, and blood vessels.

Diabetes thus corresponds to a set of metabolic disorders characterized by a persistent elevation of blood glucose levels. It results from the inability of the pancreas to produce sufficient insulin, the hormone essential for glycemic regulation, or from tissue resistance to its action.

According to the diagnostic criteria revised by the ADA (2016), the diagnosis of diabetes can be established by:

- The presence of clinical symptoms (polyuria, polydipsia, weight loss) associated with a venous plasma blood glucose ≥ 2.00 g/L (11.1 mmol/L).

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- Fasting blood glucose ≥ 1.26 g/L (7.0 mmol/L), confirmed on two occasions.
- Blood glucose at 2 hours during an oral glucose tolerance test (OGTT) ≥ 2.00 g/L (11.1 mmol/L).

It is important to note that the OGTT is no longer recommended routinely in clinical practice. Furthermore, since 2009, glycated hemoglobin (HbA1c) has also been recognized by the ADA as a diagnostic tool for diabetes. HbA1c testing every two to three months has been recognized as a method for measuring glycemic control in the care and treatment of patients with diabetes mellitus.

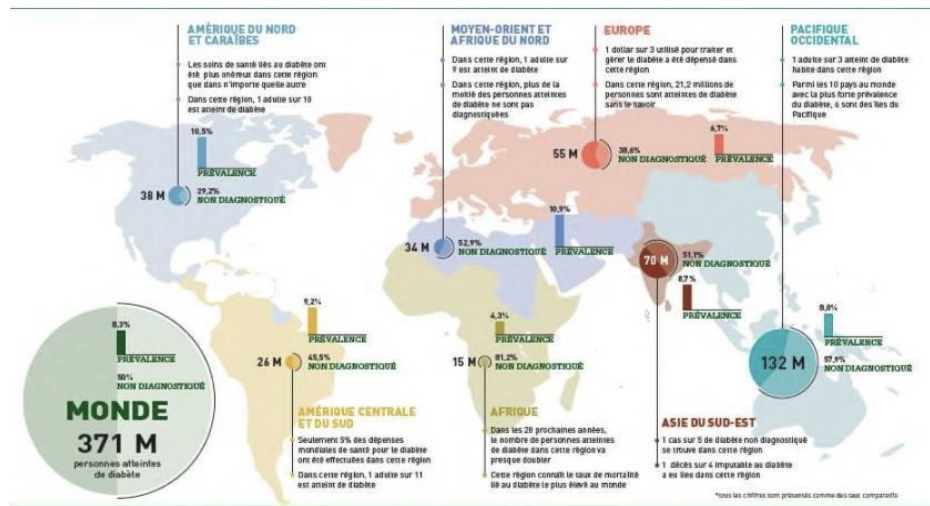
I.2.3 Epidemiology:

Diabetes is rapidly emerging as one of the greatest health crises, representing a significant financial burden due to the high rate of degenerative and cardiovascular complications (Arbouche et al., 2012). Current estimates from 2007 count 246 million people, with 5.9% of diabetics aged between 20–79 years, of which more than 70% live in emerging countries (Arbouche et al., 2012). According to the International Diabetes Federation (IDF) in 2010, 6% of the world population suffered from diabetes, and in 2012, 371 million people were diabetic worldwide. According to the World Health Organization (WHO), the prevalence would increase from 4.0% of people affected worldwide in 1995 to 5.4% in 2025 (Kebieche, 2009).

The number of deaths attributed to diabetes is around four million per year, representing 9% of total mortality (Arbouche et al., 2012). Every minute, worldwide, six people die from diabetes itself or from its complications. The WHO predicts that these deaths will increase by more than 50% over the next ten years (Kebieche, 2009).

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In the Maghreb and in Algeria, a genuine epidemiological transition is occurring, with a growing increase in chronic non-communicable diseases such as diabetes, cancers,



and cardiovascular diseases. According to the Algerian Federation of Diabetes Associations, Algeria has 2 million diabetics of all types combined (Salemi, 2010).

I.2.4 Classification of Diabetes Mellitus

I.2.4.1 Type 1 Diabetes

Formerly called insulin-dependent diabetes (IDD), it occurs following autoimmune destruction of the β cells of the islets of Langerhans, leading to a total or near-total absence of insulin secretion. This destruction is promoted by genetic and environmental factors. Type 1 diabetes mainly affects children and young adults.

I.2.4.2 Type 2 Diabetes

Formerly called non-insulin-dependent diabetes (NIDDM), it is characterized by insulin resistance associated with progressive insufficiency of insulin secretion. It mainly occurs in adults and is strongly associated with overweight and sedentary lifestyle.

I.2.4.3 Gestational Diabetes

This is glucose intolerance appearing or diagnosed for the first time during pregnancy. It is linked to physiologically increased insulin resistance during pregnancy and generally disappears after delivery, but constitutes a subsequent risk factor for type 2 diabetes.

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I.2.4.4 Other Types of Diabetes

These include secondary forms of diabetes, notably those linked to pancreatic diseases (pancreatitis, hemochromatosis), endocrine disorders (Cushing's syndrome, acromegaly), genetic abnormalities (MODY, mitochondrial diabetes), or drugs and toxic substances.

I.2.5 Pathophysiology of Type 2 Diabetes

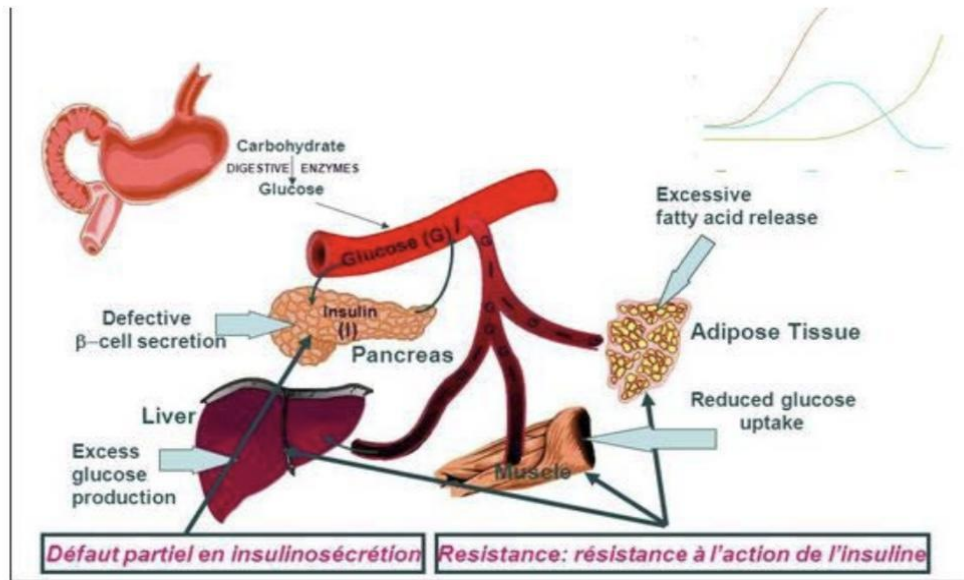
Type 2 diabetes is a chronic progressive disease whose pathophysiology begins several years before its clinical diagnosis. It is based on several interconnected metabolic mechanisms leading to progressive hyperglycemia.

The first mechanism is an alteration in the sensitivity of peripheral tissues to glucose, particularly in muscle, liver, and adipose tissue. This abnormality leads to decreased glucose utilization by muscle cells and increased hepatic glucose production, thereby contributing to elevated blood glucose levels.

The second mechanism corresponds to a progressive dysregulation of pancreatic cell function, characterized by an inability to maintain hormonal secretion adapted to the body's needs, especially in the presence of chronic hyperglycemia.

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With disease progression, a gradual decrease in the mass and functionality of pancreatic cells is also observed, partly linked to the metabolic toxicity induced by persistent



hyperglycemia. These different mechanisms lead to a progressive and lasting increase in blood glucose, responsible for the metabolic and vascular complications associated with type 2 diabetes.

This figure illustrates the main pathophysiological mechanisms involved in type 2 diabetes, including insulin resistance and impaired insulin secretion.

I.2.6 Complications

The development of diabetic complications is correlated with poor glycemic control. Each patient has their own susceptibility to develop these complications (Hennen, 2001). They are numerous, local or general, insidious, chronic, and often severe since the life expectancy of a diabetic person is reduced by five to ten years. Hyperglycemia, insulin resistance, low-grade inflammation, accelerated atherogenesis, and susceptibility to infections underlie most complications (Schlienger, 2013). They are divided into two groups: microangiopathies and macroangiopathies.

I.2.6.1 Microangiopathic Complications

Retinopathy: This is the most frequent and most specific ocular abnormality of diabetes (Bories, 2012). It is the fourth cause of visual acuity loss in diabetics over 65 years of age (Schlienger, 2013). This microangiopathy is due to the thickening of the basal membrane of retinal capillaries followed by a decrease in pericytes and a reduction in the number of endothelial cells, which leads to capillary dilation, microaneurysm formation, and retinal capillary occlusion.

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Nephropathy: Diabetes is one of the main causes of end-stage chronic renal failure. Diabetic nephropathy is primarily linked to microangiopathy and progresses through several successive stages. It affects approximately 15 to 20% of patients with type 2 diabetes (Racah, 2004; Villar and Zaoui, 2010).

Neuropathy: Diabetic neuropathy is a very frequent complication that can affect the peripheral and autonomic nervous systems (Bories, 2012). Approximately 50% of type 2 diabetics have neuropathy; its prevalence varies according to diabetes duration, quality of glycemic control, age, and association with other pathologies.

I.2.6.2 Macroangiopathic Complications

Atherosclerosis: Atherosclerosis has become the leading cause of death in diabetics over 40 years of age. It affects the heart (myocardial infarction), the brain (ischemic stroke, 2 to 5 times more frequent than in the non-diabetic population), and the lower limbs with arteritis, responsible for half of amputations.

Atheroma: Slowly progressive atheroma is characterized by the accumulation of lipids and fibrous elements in medium and large arteries, which can lead to chronic ischemic manifestations interspersed with acute atherothrombotic episodes.

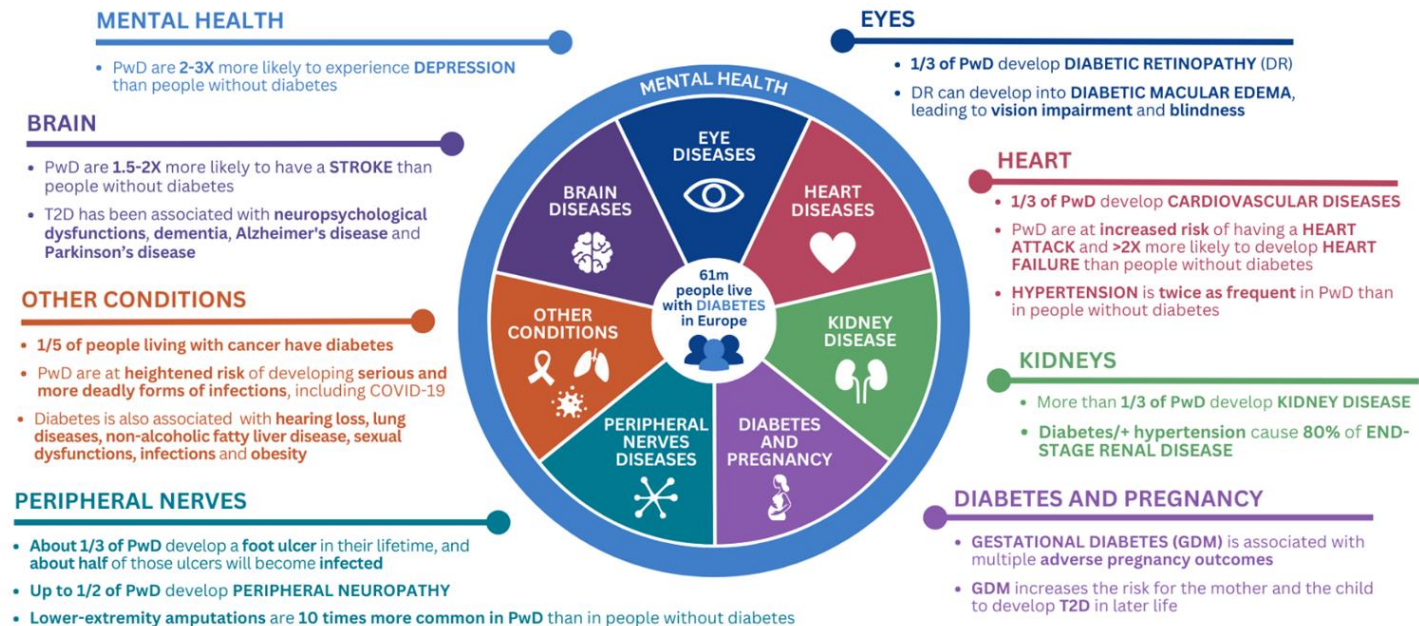
Diabetic Foot: The foot is the preferred target of diabetes. It is a serious and frequent complication as it is one of the leading causes of prolonged hospitalization (Baalbaki, 2012). Insignificant trauma can quickly turn into chronic ulcers and focal gangrene developing on tissues severely compromised by ischemic phenomena (Hennen, 2001). The high frequency of lower limb amputations proves the severity of the lesions since 85% of amputations in diabetics are preceded by a foot ulcer.

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COMPLICATIONS AND COMORBIDITIES IN PEOPLE LIVING WITH DIABETES (PWD)

Diabetes can affect every organ system in the human body. The extent of potential organ damage depends largely on its duration and successful management



IDF Europe, 2023: <https://idf.org/europe/life-with-diabetes/>

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I.2.7 Risk Factors for Type 2 Diabetes

I.2.7.1 Constitutional Factors:

- **Age and Sex:** Population aging is an additional risk factor for type 2 diabetes due to increased insulin resistance and reduced insulin secretion (Annette et al., 2003).
- **Pregnancy:** Gestational diabetes can occur from the 24th week of pregnancy and is a subsequent risk factor for type 2 diabetes.
- **Genetic Factors:** Type 2 diabetes is a polygenic disease resulting from the interaction of several genes involved in the regulation of carbohydrate metabolism, with a heritability of 60 to 100% in twin studies (Clément, 2003).

I.2.7.2 Environmental and Behavioral Factors:

- **Obesity:** Particularly abdominal obesity, is the main risk factor for type 2 diabetes (WHO, 2016).
- **Diet:** A diet rich in saturated fatty acids and high glycemic index sugars, and poor in fiber, promotes the onset of type 2 diabetes (Steyn et al., 2004).
- **Physical Inactivity:** Sedentary lifestyle is a major risk factor. Regular physical activity can reduce the risk of developing type 2 diabetes by approximately 6% in active subjects (Helmrich et al., 1991).
- **Smoking:** Smoking reduces insulin sensitivity and promotes insulin resistance through several mechanisms (Targher, 2005; Beziaud et al., 2004).
- **Sleep Deprivation:** Recent studies have shown that insufficient or poor-quality sleep can increase the risk of developing type 2 diabetes. Indeed, it leads to increased insulin resistance, hormonal imbalances regulating appetite, elevated blood glucose, weight gain, and increased obesity risk. Moreover, chronic sleep deprivation (fewer than 6 hours per night) is clearly associated with a significant increase in the risk of diabetes (Taheri et al., 2004; Cappuccio et al., 2010).

I.2.8 Therapeutic Management of Type 2 Diabetic Patients:

I.2.8.1 Insulin Therapy:

Insulin therapy consists of replacing missing insulin with daily injections of exogenous insulin, the quantity of which is determined based on blood glucose levels. Due to its

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subcutaneous administration, there is a significant delay between injection and the appearance of insulin in peripheral circulation, as well as variability in absorption from one injection to another (Klein, 2009).

I.2.8.2 Oral Antidiabetic Medications:

There are different classes of oral antidiabetics. Five of them (approved in the United States for the treatment of non-insulin-dependent diabetes mellitus in humans) are considered here: Biguanides, Glitazones, Sulfonylureas, Glinides, and Alpha-glucosidase inhibitors. The first two reduce insulin resistance, while the last three stimulate insulin secretion (Klein, 2009).

I.2.8.3 Lifestyle Modifications and Physical Exercise:

The goal of dietary management is not to deprive the diabetic patient of life's pleasures, but to avoid carbohydrate intake that is not adapted. A varied and balanced diet, in respect of the most regular possible eating rhythm, is recommended. Physical activity is essential to combat the chronic hyperglycemia of well-controlled diabetes mellitus and delays the appearance of certain vascular complications. However, it is strongly discouraged for uncontrolled diabetes as it increases the risk of hyperglycemia and ketosis due to insulin deficiency.

In this context, type 2 diabetes represents a complex multifactorial pathology, whose continued global progression constitutes a major public health challenge. Its management relies on a comprehensive understanding of pathophysiological mechanisms and associated risk factors. Among these factors, lifestyle-related determinants occupy a central place, including diet, physical activity, and, more recently, sleep quality. Identifying these behavioral factors opens important prospects for the prevention and improvement of glycemic control in patients with type 2 diabetes.

I.2.9 Relationship between sleep and type 2 diabetes:

Among the lifestyle-related factors involved in the development and management of type 2 diabetes, sleep has recently emerged as an important determinant of metabolic health. Growing evidence suggests that inadequate sleep duration and poor sleep quality are associated with an increased risk of type 2 diabetes. Sleep disturbances may alter glucose metabolism, impair insulin action, and contribute to metabolic dysregulation, thereby favoring the development and progression of the disease. Consequently, adequate and restorative sleep is increasingly recognized as a key component of strategies aimed at preventing type 2 diabetes and improving glycemic control (Shan et al., 2015).

I.2.10 Chapter summary

Taken together, these findings highlight the multifactorial nature of type 2 diabetes and emphasize the importance of understanding the underlying mechanisms involved in its

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development. Among these mechanisms, insulin resistance occupies a central role and represents a major contributor to the onset and progression of the disease.

CHAPTER 3: INSULIN RESISTANCE

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I.3.1 Introduction:

Insulin resistance is a central element in the pathophysiology of metabolic disorders, particularly type 2 diabetes. It is defined as a decrease in the sensitivity of peripheral tissues to the action of insulin, the hormone essential for glycemic regulation (DeFronzo, 2004).

Under normal conditions, insulin allows glucose to enter cells, particularly in muscle and adipose tissue, while inhibiting hepatic glucose production. However, in a situation of insulin resistance, these mechanisms are altered, leading to an elevation of blood glucose levels (Saltiel & Kahn, 2001).

To compensate for this anomaly, the body increases insulin secretion, leading to transient hyperinsulinemia. In the long term, this compensation becomes insufficient due to the progressive exhaustion of pancreatic β cells, thereby promoting the onset of type 2 diabetes (Prentki & Nolan, 2006).

Several factors contribute to the development of insulin resistance, including obesity, sedentary lifestyle, genetic factors, and sleep disturbances. Indeed, sleep deprivation and circadian rhythm disorders play an important role in altering insulin sensitivity by modifying hormonal and metabolic balance (Spiegel et al., 2005).

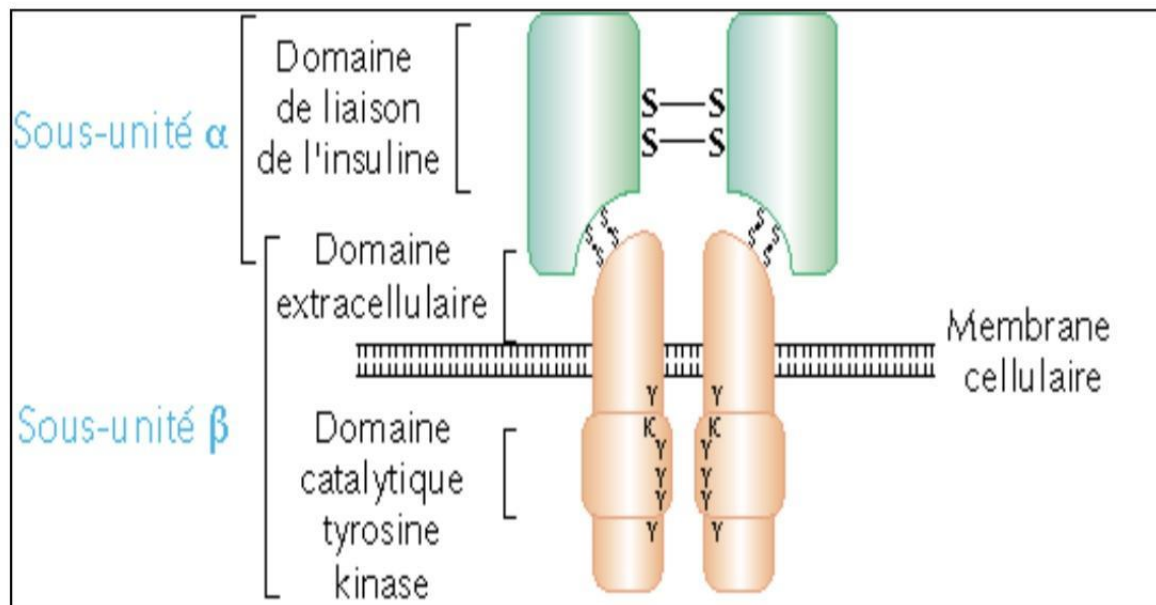
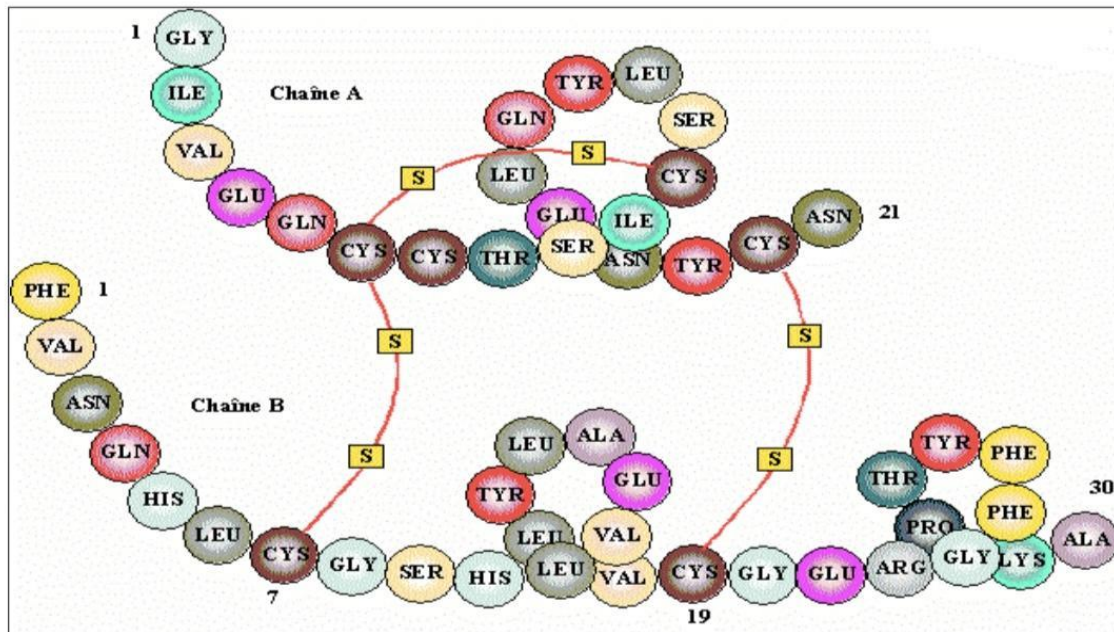
I.3.2 General Overview of Insulin:

I.3.2.1 Insulin and Its Receptor:

Insulin is a small protein whose 51 amino acids are produced and secreted by the β cells of the pancreatic islets. It consists of two polypeptide chains, α and β , of 21 and 30 amino acids respectively, which are connected by disulfide bridges (-S-S-) (Marieb and Hoehn, 2015; Arnold et al., 2018). It is synthesized as a long polypeptide chain called proinsulin, from which enzymes cleave the middle portion, releasing insulin (Marieb and Hoehn, 2015). Insulin acts as a hypoglycemic hormone, and possesses major anabolic activity in the storage of carbohydrate and lipid substrates (Benaraba, 2007).

The insulin receptor belongs to the family of IGF1 (Insulin-Like Growth Factor 1) growth factor receptors that possess tyrosine kinase activity in their intracellular domain. It is an integral membrane glycoprotein, formed of four subunits: two extracellular α subunits linked by disulfide bridges and two transmembrane β subunits (Marieb and Hoehn, 2015). Insulin binding to the α subunits causes a conformational change in the insulin receptor and consequently induces autophosphorylation of several Tyr residues located in the cytosolic region of the β subunit, then the phosphorylation of tyrosine residues of substrate proteins, IRS proteins (Insulin Receptor Substrates), among which IRS-1 and IRS-2 are the two main substrates and the most common intermediaries in the initial stage of insulin signal propagation (Arnold et al., 2018).

CHAPTER 3: INSULIN RESISTANCE



CHAPTER 3: INSULIN RESISTANCE

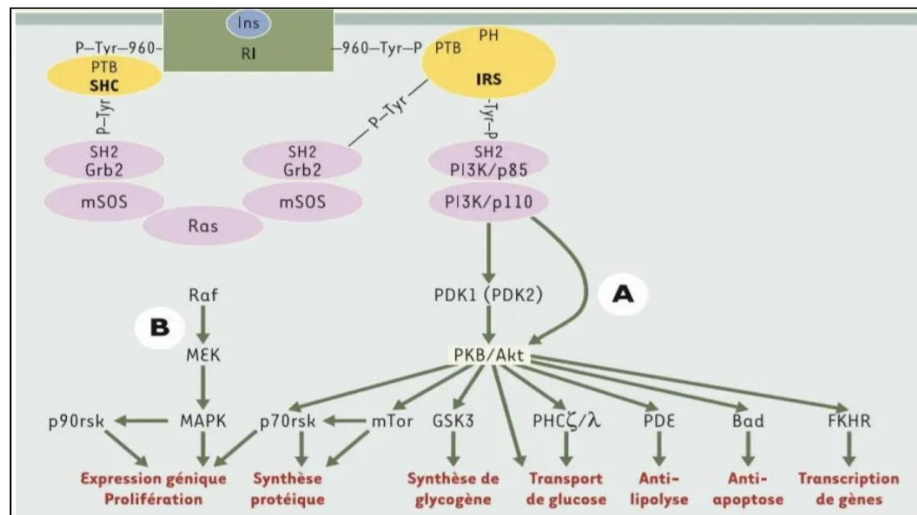
I.3.2.2 Insulin Signaling:

Activation of the insulin receptor and phosphorylation of IRS substrates are essential for signal transduction. IRS proteins 1 to 4 can be involved in signal transduction, but IRS 1 and IRS 2 are the most important for glucose transport. Phosphorylated IRS proteins recruit molecules containing SH-2 (Src-homology-2) domains such as phosphatidylinositol 3-kinase (PI3K), GRB-2/mSOS, and SHP-2. Several signaling pathways induced by insulin are distinguished, including the PI3K pathway and the mitogen-activated protein kinase (MAPK) pathway (Tan-Chen et al., 2020).

PI3K is the main element of insulin-dependent metabolic actions. The p85 regulatory subunit of PI3K binds the phosphorylated tyrosine residues of IRS, which leads to increased catalytic activity of the p110 subunit, which phosphorylates PtdIns(4,5)P₂ at position 3 to give PtdIns(3,4,5)P₃. PtdIns(3,4,5)P₃ recruits kinases PDK-1, PKB/Akt, and PKC, which are involved in different insulin-related responses such as GLUT4 translocation to the plasma membrane, glycogen synthesis via phosphorylation of glycogen synthase kinase-3, and lipogenesis. Akt therefore acts as a major relay for the metabolic actions of insulin in its target tissues (Tan-Chen et al., 2020).

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The MAPK signaling pathway leads to the expression of genes involved in cellular differentiation and proliferation. After IRS phosphorylation, GRB-2 and SOS proteins are recruited and act with SHP-2 to activate Ras, an intermediate protein in the phosphorylation cascade leading to ERK (Extracellular signal-regulated kinase) recruitment and ultimately the phosphorylation of transcription factors activating the expression of different genes. The growth-promoting and gene-regulatory effects of insulin are mainly mediated through activation of the MAPK/Ras pathway (Capeau, 2003; Arnold et al., 2018).



I.3.2.3 Negative Regulation of Insulin Action:

Insulin signaling is negatively regulated by several mechanisms, including hormone degradation as well as recycling or degradation of its receptors. Under physiological conditions, the body maintains a sufficient number of insulin receptors through their neosynthesis. However, in cases of chronic hyperinsulinism, this can lead to a decrease in the number of receptors on the cell surface (down-regulation), thereby contributing to the development of insulin resistance (Capeau, 2003).

Furthermore, the arrest of intracellular signaling can occur through dephosphorylation of the insulin receptor and IRS proteins through the action of phosphatases. An increase in the activity of these enzymes has been observed in the muscles of diabetic patients, which reinforces the state of insulin resistance (Capeau, 2003).

I.3.2.4 Involvement of Insulin in Metabolism:

Insulin plays an essential role in regulating carbohydrate, lipid, and protein homeostasis. It acts primarily at the level of skeletal muscle, liver, adipose tissue, and the cardiovascular system (Saltiel and Kahn, 2001).

CHAPTER 3: INSULIN RESISTANCE

In terms of carbohydrate metabolism, insulin promotes glucose entry into insulin-sensitive cells, particularly through the translocation of the GLUT4 transporter to the plasma membrane. It also stimulates glucose utilization and storage in the form of glycogen in the liver and muscle while inhibiting hepatic glucose production (Tan-Chen et al., 2020).

Regarding lipid metabolism, insulin promotes lipid storage by stimulating lipogenesis and lipoprotein lipase activity while inhibiting free fatty acid mobilization. At the protein level, it increases amino acid uptake and stimulates protein synthesis through intracellular signaling pathways (Saltiel and Kahn, 2001; Tan-Chen et al., 2020).

Finally, insulin also exerts cardiovascular effects, notably a vasodilatory action through nitric oxide (NO) production, modulation of vascular and cardiac contractility, and a role in microcirculation (Muniyappa et al., 2007).

I.3.3 Insulin Resistance

I.3.3.1 Definition:

Insulin resistance is defined as a decrease in the response of target tissues to the action of insulin, particularly skeletal muscle, cardiac muscle, adipose tissue, and liver. This anomaly is generally compensated by hypersecretion of insulin leading to hyperinsulinemia. It constitutes a major pathophysiological mechanism involved in the development of type 2 diabetes, which combines both an alteration in insulin sensitivity and a progressive deficit in its secretion (Scheen, 2001; Ormazabal et al., 2018).

Insulin resistance can be considered a pre-diabetic state that remains asymptomatic for a long time. The mechanisms responsible for the progressive loss of insulin sensitivity may occur at different levels of insulin metabolism, including the insulin receptor itself and downstream signaling pathways (Scheen, 2001).

I.3.3.2 Prevalence:

The prevalence of insulin resistance is difficult to determine precisely, due to the absence of a universally accepted reference method for its evaluation. Estimates from 2007 showed that approximately 197 million people worldwide had glucose intolerance, considered an indirect indicator of insulin resistance. In the United States, approximately 25% of the population is affected, representing about 60 million people. Projections indicate a significant increase in this prevalence, potentially reaching approximately 420 million people with glucose intolerance in 2025 (Hossain, 2007).

I.3.3.3 Stages of Insulin Resistance Progression :

1. **Initial Phase:** Characterized by insulin resistance compensated by hyperinsulinemia, maintaining normal fasting blood glucose. Insulin secretion progressively increases to compensate for the decreased tissue sensitivity to insulin (Scheen, 2001).

CHAPTER 3: INSULIN RESISTANCE

2. **Alteration of Hepatic Glucose Production:** After a prolonged period of hyperinsulinism, compensatory mechanisms become insufficient, leading to increased hepatic glucose production and the gradual onset of hyperglycemia (Scheen, 2001).
3. **Failure of Insulin Secretion:** At an advanced stage, exhaustion of pancreatic β cells leads to insulinopenia, making insulin therapy necessary. This stage is accompanied by a decreased ability of insulin to inhibit hepatic glucose production and promote peripheral utilization (Scheen, 2001; Godsland, 2010).

I.3.3.4 Molecular Mechanisms of Insulin Resistance:

Disruptions in the insulin signaling pathway are responsible for the development of insulin resistance (Scheen, 2001; Szymczak-Pajor and Śliwińska, 2019). These disruptions are caused by various alterations, notably mutations and/or post-translational modifications of the insulin receptor, IRS proteins, or downstream effector molecules. The most common insulin resistance alterations include: decreased number of insulin receptors through accelerated receptor degradation associated with the hormone; decreased receptor catalytic activity; reduced tyrosine phosphorylation of IRS1; decreased PI3-kinase activation; impaired GLUT4 translocation; reduced glucose transport; decreased glucose phosphorylation; diminished glycolytic flux and glucose oxidation; and impaired glycogen synthesis (Clauser, 1988; Ormazabal et al., 2018).

I.3.3.5 Causes of Insulin Resistance:

I.3.3.5.1 Lifestyle Factors:

- **Unbalanced diet:** Excessive consumption of fats and refined sugars disrupts glucose metabolism and promotes insulin resistance (Fève et al., 2006).
- **Vitamin D deficiency:** Low vitamin D levels can impair insulin secretion as well as cellular sensitivity to this hormone, thereby compromising glucose tolerance (Ntyonga-Pono, 2014).
- **Overweight and obesity:** Obesity, particularly visceral fat accumulation, constitutes a major factor of insulin resistance. Adipose tissue secretes various substances and hormones (such as leptin and adiponectin) influencing insulin sensitivity (Petersen and Shulman, 2006; Ye, 2013).
- **Sleep deprivation and sleep disorders:** Recent studies have shown that insufficient or poor-quality sleep leads to hormonal imbalances, particularly increased cortisol secretion and disruption of appetite-regulating hormones. These alterations contribute to reduced insulin sensitivity and elevated blood glucose levels. In addition, sleep deprivation is associated with increased inflammation and oxidative stress, which further promote the development of insulin resistance. Therefore, adequate sleep duration and good sleep quality are essential for maintaining metabolic homeostasis (Spiegel et al., 2002; Liu et al., 2022).

I.3.3.5.2 Genetic Factors :

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- Mutations in genes involved in insulin signaling, notably IRS-1 and IRS-2 (Ormazabal et al., 2018).
- The presence of family history of type 2 diabetes, increasing individual susceptibility (Ye, 2013).

I.3.3.6 Evaluation of Insulin Resistance:

Several indices have been developed to estimate insulin resistance. Among them, the HOMA-IR (Homeostatic Model Assessment for Insulin Resistance) index is the most commonly used. It is based on the relationship between blood glucose and insulinemia, according to the following formula: $(\text{blood glucose [mmol/l]} \times \text{insulinemia [mIU/l]}) / 22.5 > 1.8\text{--}2.0$. A low HOMA-IR index indicates high insulin sensitivity, while a high HOMA-IR index indicates low insulin sensitivity. $\text{HOMA-IR} > 4.4$ has been considered compatible with insulin resistance (Stagi et al., 2017).

I.3.3.7 Prevention and Management of Insulin Resistance:

Given the increasing prevalence of insulin resistance worldwide, the implementation of preventive strategies is essential. The most effective approaches are based on lifestyle modification (Zaccaria et al., 2025; American Diabetes Association, 2024):

- **Weight management:** Achieving and maintaining a healthy body weight, particularly through weight reduction in overweight or obese individuals, improves insulin sensitivity and reduces the risk of type 2 diabetes (American Diabetes Association, 2024).
- **Reduction of sedentary behavior:** Limiting prolonged sitting time and increasing daily movement contribute to better metabolic health and insulin action (Zaccaria et al., 2025).
- **Regular physical activity:** Engaging in at least 150 minutes of moderate-intensity physical activity per week improves glucose uptake and insulin sensitivity (American Diabetes Association, 2024; Pedram et al., 2021).
- **Adequate sleep:** Maintaining sufficient sleep duration and quality helps preserve hormonal balance and metabolic function, thereby reducing the risk of insulin resistance (Liu et al., 2022; Zaccaria et al., 2025).
- **Balanced diet:** Consuming a diet rich in fruits, vegetables, whole grains, and healthy fats while reducing the intake of refined sugars and saturated fats contributes to improved insulin sensitivity (Zaccaria et al., 2025; American Diabetes Association, 2024).
- **Regular medical follow-up:** Monitoring blood glucose levels and metabolic parameters allows early detection and management of insulin resistance and related complications.

I.3.3.8 Acanthosis Nigricans as an Early Marker of Insulin Resistance:

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Insulin resistance may manifest clinically through several early cutaneous signs. One of the most common is skin darkening in the neck region, known as **acanthosis nigricans**. This condition is characterized by hyperpigmented, thickened, velvety plaques that typically develop in skin folds, particularly the neck, axillae, and groin (StatPearls, 2023; Antonio Videira-Silva et al., 2019).

The condition is primarily associated with hyperinsulinemia, which stimulates keratinocyte proliferation and melanocyte activity, leading to skin thickening and hyperpigmentation (StatPearls, 2023). Consequently, acanthosis nigricans is considered an early clinical marker of impaired insulin sensitivity, especially in overweight individuals and those at increased risk of developing type 2 diabetes (Antonio Videira-Silva et al., 2019; Herman et al., 1986).

I.3.3.9 Chapter summary

Recognition of this clinical sign is important because it facilitates early diagnosis and the implementation of preventive interventions, including lifestyle modification, weight reduction, and regular physical activity, which can improve insulin sensitivity and reduce metabolic risk (Antonio Videira-Silva et al., 2019).

Overall, this chapter highlights that understanding the mechanisms and determinants of type 2 diabetes, particularly insulin resistance and lifestyle-related factors, is essential for guiding effective preventive strategies.

PART II: PRACTICAL PART

CHAPTER 4:MATERIALS AND METHODS

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Study: Sleep Deprivation as a Contributing Factor to Insulin Resistance and Type 2 Diabetes

II.4.1 Type and Design of the Study:

This study adopts a cross-sectional descriptive and analytical design. The approach is entirely quantitative, relying on structured self-administered questionnaires distributed to two distinct population groups at a single point in time. This design was deliberately chosen because it allows simultaneous exploration of sleep-related exposures and metabolic outcomes without any experimental intervention. It is cost-effective, feasible within an academic research framework, and well-suited to generating meaningful associations between variables that may later be confirmed through longitudinal or interventional studies.

II.4.2 Research Objectives:

The general objective of this study is to evaluate the relationship between sleep deprivation and the risk of insulin resistance and type 2 diabetes in university students, while simultaneously assessing the impact of sleep quality on glycemic control in patients already diagnosed with type 2 diabetes.

To achieve this general objective, five specific objectives were defined. The first is to describe the sleep duration and quality profiles of university students enrolled in health-science programs. The second is to identify the proportion of students presenting metabolic risk indicators based on their lifestyle habits and anthropometric measurements. The third is to assess sleep patterns and nocturnal disturbances in type 2 diabetic patients. The fourth is to examine the self-perceived relationship between sleep quality and glycemic control in the diabetic population. The fifth and final specific objective is to validate four operational hypotheses linking sleep deprivation to distinct dimensions of metabolic dysregulation.

II.4.3 Research Hypotheses:

The study is structured around four research hypotheses derived from the current scientific literature on sleep and metabolic health. The first hypothesis states that a sleep duration of fewer than seven hours per night may be associated with an increased risk of glycemic imbalance. The second hypothesis proposes that poor sleep quality may lead to hormonal disturbances, notably an increase in cortisol secretion and alterations in growth hormone release, both of which are critical regulators of insulin sensitivity. The third hypothesis suggests that irregular sleep habits — such as late bedtimes and reduced sleep duration — may be common among university students and may negatively influence their metabolic balance. The fourth hypothesis posits that in patients already diagnosed with type 2 diabetes, sleep deprivation may be associated with less optimal glycemic control, as reflected by elevated HbA1c values and increased treatment burden.

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II.4.4 Study Population:

The study targets two distinct populations. The first population consists of university students enrolled in health-science disciplines — specifically Biology, Medicine, Nursing, and Nutrition — representing a young adult group exposed to academic stress, irregular schedules, and lifestyle-driven sleep disruption. The second population consists of adults with a confirmed diagnosis of type 2 diabetes, recruited from healthcare facilities, representing a clinical population with established metabolic disease and an ongoing relationship with the healthcare system.

A sample of 98 participants was recruited for each group, yielding a total sample of 196 participants across both questionnaires. This sample size was determined to be sufficient for descriptive and comparative statistical analyses within the constraints of an academic research setting. The sampling method employed was convenience sampling, a non-probabilistic approach selected for its feasibility and accessibility given the study's timeframe and resources.

Inclusion criteria for the student group required participants to be actively enrolled in a health-science program, aged 18 years or above, free of any prior diagnosis of diabetes or insulin resistance, and willing to provide voluntary informed consent. For the diabetic patient group, inclusion required a confirmed diagnosis of type 2 diabetes supported by medical records or treating physician confirmation, a disease duration of at least six months, current pharmacological treatment with oral antidiabetics, insulin, or a combination of both, and voluntary informed consent.

Exclusion criteria were applied uniformly across both groups and included pregnancy or breastfeeding, a diagnosis of type 1 or secondary diabetes, the presence of severe psychiatric disorders known to affect sleep architecture, the ongoing use of sleep-altering medications unrelated to diabetes management, and incomplete questionnaire responses with more than 20% missing data.

II.4.5 Data Collection Tool:

Data were collected using two structured self-administered questionnaires developed specifically for this study. Both instruments were designed drawing upon internationally validated tools, particularly the Pittsburgh Sleep Quality Index (PSQI) and the Epworth Sleepiness Scale (ESS), adapted to the specific objectives and population characteristics of this research.

The first questionnaire, addressed to university students, comprises 14 items organized across six thematic sections. The first section collects sociodemographic information including age, sex, level of study, field of study, and family history of diabetes. The second section addresses sleep duration through questions on average nightly sleep, usual bedtime, and wake-up time. The third section evaluates sleep quality by exploring the frequency of difficulty falling asleep and the number of nighttime awakenings. The fourth section examines lifestyle factors, specifically physical activity level and daily caffeine consumption. The fifth section records anthropometric data through self-reported body weight. The sixth and final section collects

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metabolic information, asking whether the participant has ever undergone blood glucose testing and, if so, the reported value.

The second questionnaire, addressed to type 2 diabetic patients, comprises 13 items also organized across six sections. The first section gathers clinical data including age, sex, duration of diabetes, type of treatment, and the most recent HbA1c value. The second section assesses nightly sleep duration. The third section evaluates sleep quality through questions on sleep onset difficulty and nighttime awakening frequency. The fourth section explores the perceived relationship between sleep and glycemic control, asking whether blood sugar appears higher after a poor night's sleep, whether hunger increases following sleep deprivation, and what the main causes of nocturnal awakenings are. The fifth section examines the frequency of self-monitoring of blood glucose. The sixth section records body weight as an anthropometric indicator.

The questionnaires were administered individually or in small groups on university campuses and at affiliated healthcare facilities. Prior to distribution, all participants received a verbal and written explanation of the study's objectives, voluntary nature, and confidentiality guarantees. The questionnaires were self-administered and anonymous, and were collected immediately upon completion to minimize non-response and data loss. The average time required to complete each questionnaire was estimated at between eight and twelve minutes.

II.4.6 Variables Studied:

The independent variables — representing the exposure of interest — include sleep duration expressed in hours per night, sleep quality operationalized through self-reported difficulty falling asleep and frequency of nighttime awakenings, and sleep schedule regularity reflected by bedtime and wake-up time. The dependent variables — representing the metabolic outcomes of interest — include HbA1c level as the primary marker of long-term glycemic control, self-reported blood glucose values, and the patient's perceived link between sleep quality and blood sugar levels. Several confounding and moderating variables were also measured, including physical activity level, daily caffeine consumption, body weight, family history of diabetes, and, in the diabetic group, treatment type and disease duration. These variables were included because they are known to independently influence both sleep quality and metabolic outcomes, and their effects must be considered when interpreting associations between sleep and glycemic indicators.

II.4.7 Ethical Considerations

This study was conducted in strict adherence to ethical research principles. Prior to data collection, official authorization was obtained from the university (Dr. Moulay Tahar University, Saida) and permission to conduct the study was granted by the hospital administration (Sadouki Abdelkader Public Hospital, Sidi Boubekeur, Saïda, Algeria) . All participants were fully informed of the study's objectives and provided voluntary consent. Anonymity and confidentiality were guaranteed throughout the research process. Participants retained the right to withdraw at any time without consequence. No harmful procedures were employed, and all data were used solely for scientific purposes.

CHAPTER 5: RESULTS

ANALYTICAL PHASE

QUESTIONNAIRE 01 – University Students: Sleep & Risk of Insulin Resistance

II.5.1 Sociodemographic Data

Question 01: Age

Table 01: Age Distribution of Participants

Age Group	Count	Percentage
18–30 years	50	51,0%
30-40 years	38	38,8%
40-50 years	10	10,2%
Total	98	100%

Table 1: Age Distribution of Participants

Analysis:

The majority of participants were aged 18–30 years (51%, n=50), followed by 30–40 years (39%, n=38), and 40–50 years (10%, n=10), with the sample skewing predominantly toward young adults under 40 (90%).

Interpretation:

The predominance of younger participants suggests a student or early-career population particularly exposed to lifestyle-related risk factors such as irregular sleep, sedentary behavior, and poor diet — all central to this study. The limited representation of the 40–50 age group (10%) is acknowledged as a limitation, as metabolic risk increases significantly with age.

Question 02: Sex

Table 02: Distribution by Sex

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Sex	Count	Percentage
Female	50	51%
Male	48	49%
Total	98	100%

Table 2: Distribution by Sex

Analysis:

The sample is nearly equally split: 51% female and 49% male, with a difference of only 2 participants.

Interpretation:

The near-equal sex distribution ensures balanced representation and reduces sex-related bias in the findings. This balance is favorable for comparing sleep and metabolic outcomes across sexes.

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Question 03: Level of Study

Table 03: Distribution by Academic Level

Level of Study	Count	Percentage
Bachelor's	37	37,8%
Master's	40	40,8%
Doctorate	21	21,4%
Total	98	100%

Table 3: Distribution by Academic Level

ANALYTICAL PHASE

Analysis:

Master's students make up the largest group (40%), followed by Bachelor's (37%) and Doctorate students (21%). One participant's data was missing (n=98).

Interpretation:

The dominance of Master's and Bachelor's students reflects a typical university demographic. Higher academic levels often correlate with greater academic stress and irregular sleep schedules, which may amplify insulin resistance risk.

Question 04: Field of Study

Table 04: Distribution by Field of Study

Field	Count	Percentage
Biology	34	34,7%
Medicine	31	31,6%
Nursing	28	28,6%
Nutrition	5	5,1%
Total	98	100%

Table 4: Distribution by Field of Study

ANALYTICAL PHASE

Analysis:

Health-science fields dominate: Biology (34,7%) Medicine (31,6%) , Nursing (28,6%)and Nutrition (5,1%) All participants come from health-related disciplines.

Interpretation:

Recruiting exclusively from health sciences ensures participants have baseline awareness of metabolic health, which may influence self-reported data quality. The underrepresentation of nutrition students (5%) is notable given their field's direct relevance to the study topic.

Question 05: Family History of Diabetes

Table 05: Prevalence of Family History of Diabetes

Family History	Count	Percentage
Yes	59	60%
No	39	40%
Total	98	100%

Table 5:Prevalence of Family History of Diabetes

ANALYTICAL PHASE

Analysis:

60% of participants report a positive family history of diabetes, compared to 40% who do not.

Interpretation:

A majority having a genetic predisposition significantly heightens the relevance of the study. Participants with family history are at higher baseline risk for insulin resistance, making sleep deprivation an even more critical modifiable risk factor in this population.

II.5.2 Sleep Duration

Question 06: Average Sleep Duration per Night

Table 06: Distribution of Nightly Sleep Duration

Sleep Duration	Count	Percentage
< 5 h	5	5,1%
5–6 h	10	10,2%
6–7 h	26	26,5%
7–8 h	45	45,9%
> 8 h	12	12,2%
Total	98	100%

ANALYTICAL PHASE

Table 6: Distribution of Nightly Sleep Duration

Analysis:

The majority of participants slept 7–8 hours per night (45.9%), followed by 6–7 hours (26.5%). A smaller proportion (15.3%) reported sleeping less than 6 hours, while 12.2% slept more than 8 hours per night. This suggests that most participants achieved a sleep duration within the recommended range.

Interpretation:

With one-third of students sleeping below the recommended 7-hour threshold, this population faces elevated risk of sleep-deprivation-induced metabolic disruption. Short sleep duration is directly linked to impaired glucose tolerance and increased insulin resistance.

Question 07: Bedtime

Table 07: Distribution of Bedtime

Bedtime	Count	Percentage
20:00	26	26,5%
21:00	25	25,5%
22:00	24	24%
23:00	23	23%
Total	98	100%

Table 7: Distribution of Bedtime

ANALYTICAL PHASE

Analysis:

Bedtimes are almost uniformly distributed across 20:00–23:00 (23–26% each), suggesting no single dominant bedtime pattern.

Interpretation:

The uniform spread of bedtimes suggests varied chronotypes (morning vs. evening types) in the sample. Late bedtimes (22:00–23:00) combined with early wake-up times can result in chronic sleep restriction, increasing circadian rhythm disruption and metabolic risk.

Question 08: Wake-up Time

Table 08: Distribution of Wake-up Time

Wake-up Time	Count	Percentage
06:00	39	39,8%
07:00	59	60,2%
Total	98	100%

Table 8: Distribution of Wake-up Time

ANALYTICAL PHASE

Analysis:

The majority wake up at 07:00 (60%), while (40%) wake at 06:00. No participants reported waking later than 07:00.

Interpretation:

Early wake-up times, particularly for students who sleep after 22:00–23:00, result in sleep durations of 7 hours or less. This pattern aligns with the sleep duration data showing a significant proportion sleeping fewer than recommended hours.

II.5.3 Sleep Quality

Question 09: Difficulty Falling Asleep

Table 09: Frequency of Difficulty Falling Asleep

Response	Count	Percentage
Never	48	49,0%
Sometimes	24	24,5%
Often	26	26,5%
Total	98	100%

Table 9:Frequency of Difficulty Falling Asleep

ANALYTICAL PHASE

Analysis:

While 49% report never having difficulty falling asleep, 51% do — 24% sometimes and 27% often. More than a quarter of students frequently struggle to initiate sleep.

Interpretation:

Sleep onset difficulties are a hallmark of insomnia and poor sleep quality. The 27% who often struggle are at high risk of chronic sleep deprivation, which directly affects glucose metabolism through elevated cortisol and impaired insulin signaling.

Question 10: Nighttime Awakenings

Table 10: Frequency of Nighttime Awakenings

Awakenings	Count	Percentage
None	27	27,6%
1–2	49	50,0%
>2	22	22,4%
Total	98	100%

Table 10: Frequency of Nighttime Awakenings

ANALYTICAL PHASE

Activity Level	Count	Percentage
Sedentary	29	29,6%
Moderate	41	41,8%
Regular	28	28,6%
Total	98	100%

Analysis:

72% of students experience at least one nighttime awakening: 50% wake 1–2 times and 22% wake more than twice. Only 28% report uninterrupted sleep.

Interpretation:

Fragmented sleep is a strong independent predictor of insulin resistance. Even when total sleep time is adequate, frequent awakenings reduce deep (slow-wave) sleep, which is critical for growth hormone release and glucose regulation.

II.5.4 Lifestyle factors

Question 11: Physical Activity Level

Table 11: Distribution of Physical Activity Level

ANALYTICAL PHASE

Table 11: Distribution of Physical Activity Level

Analysis:

42% have moderate activity, 30% are sedentary, and only 29% exercise regularly. Combined, 72% do not maintain regular physical activity.

Interpretation:

Physical inactivity significantly amplifies insulin resistance risk. With 72% lacking regular exercise, this population is doubly vulnerable — insufficient sleep combined with low physical activity creates a compounding metabolic burden.

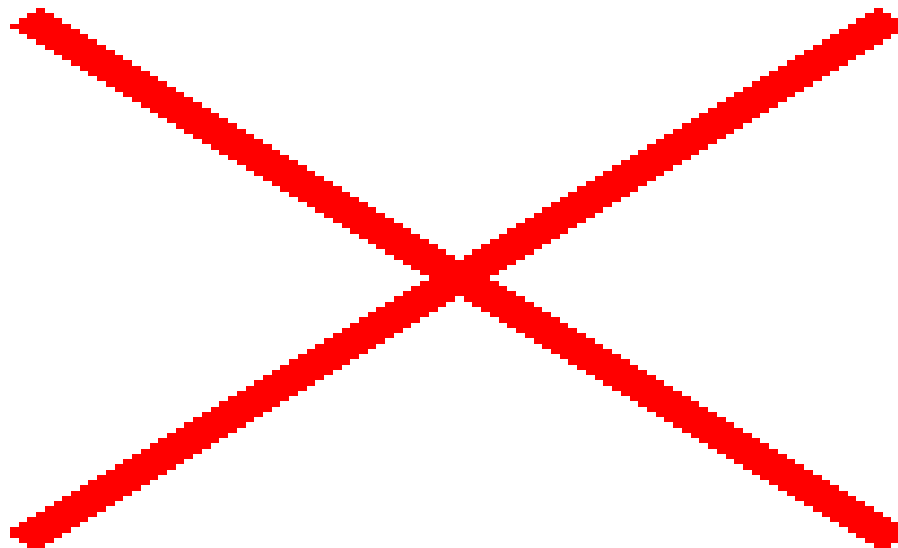
Question 12: Caffeine Consumption

Table 12: Daily Caffeine Consumption Frequency

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Caffeine Intake	Count	Percentage
None	25	25,5%
1–2/day	43	43,9%
>3/day	30	30,6%
Total	98	100%

Table 12: Daily Caffeine Consumption Frequency



Analysis:

74% consume caffeine daily: 44% consume 1–2 drinks/day and 31% consume more than 3. Only 26% abstain entirely.

ANALYTICAL PHASE

Interpretation:

High caffeine intake, especially in the evening, is a known disruptor of sleep onset and quality. The 31% consuming >3 caffeinated drinks daily are particularly at risk for delayed sleep, reduced total sleep time, and subsequent metabolic consequences.

II.5.5

Weight	Count	Percentage
65 kg	21	21,4%
85 kg	33	33,7%
90 kg	44	44,9%
Total	98	100%

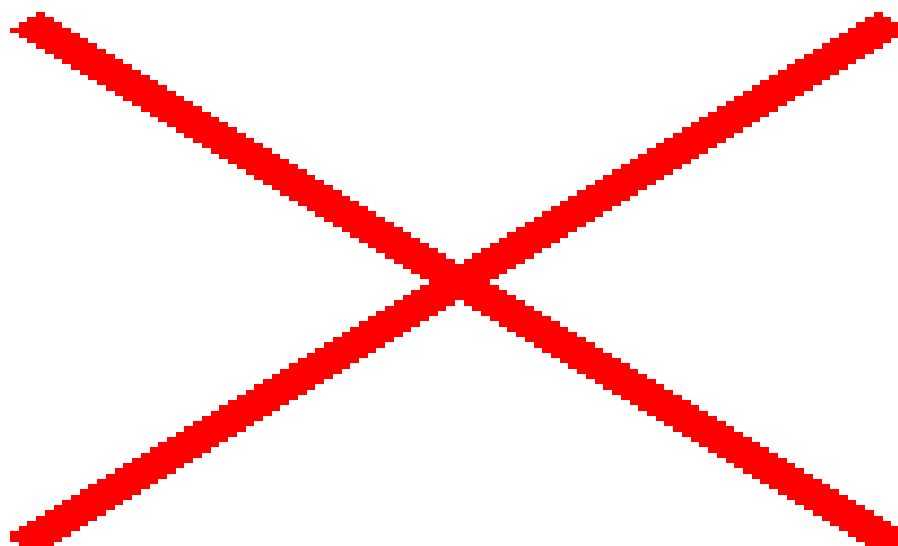
Anthropometric Measurements

Question 13: Body Weight

Table 13 : Body Weight Distribution

*Table
Weight*

13:Body



ANALYTICAL PHASE

Distribution

Analysis:

45% of participants weigh 90 kg, 34% weigh 85 kg, and only 21% weigh 65 kg. The majority fall in the overweight-to-obese range (85–90 kg).

Interpretation:

High body weight (85–90 kg) in a predominantly older student population strongly correlates with increased visceral adiposity and insulin resistance. Excess weight is both a cause and consequence of sleep disruption, creating a bidirectional risk cycle.

II.5.6 Section 6: Metabolic Data

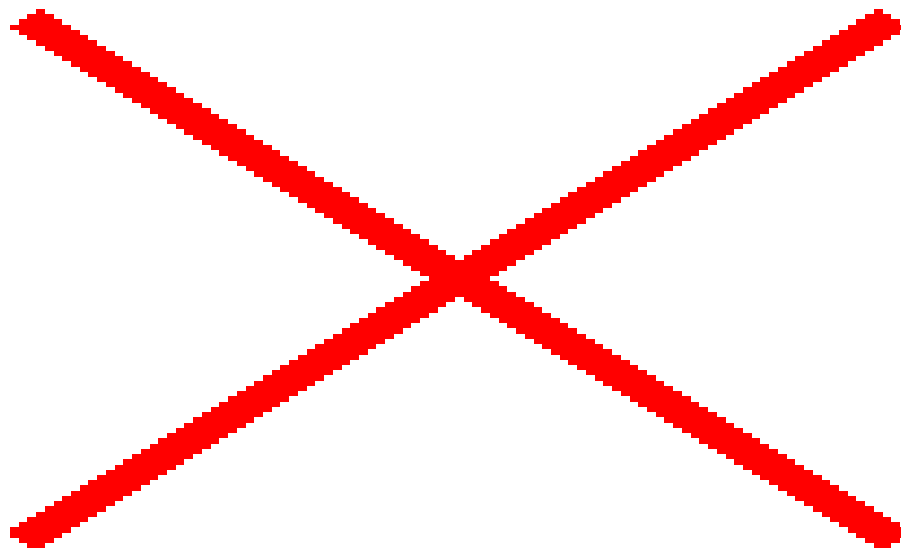
Question 14: Blood Sugar Tested

Table 14: Proportion Who Have Tested Blood Sugar

Tested	Count	Percentage
Yes	53	54,1%
No	45	45,9%
Total	98	100%

Table 14:Proportion Who Have Tested Blood Sugar

ANALYTICAL PHASE



Analysis:

54% of students have had their blood sugar tested at least once, while 46% have never done so.

Interpretation:

Nearly half the sample has never been screened for blood glucose despite many having family history of diabetes and risk factors like poor sleep and excess weight. This gap in screening highlights a critical public health concern, particularly given that early detection of prediabetes enables preventive intervention.

Questionnaire 02 – Type 2 Diabetic Patients Recruited from the Hospital Diabetes Service:

Sleep Quality & Glycemic Control

II.5.7 Clinical Data

ANALYTICAL PHASE

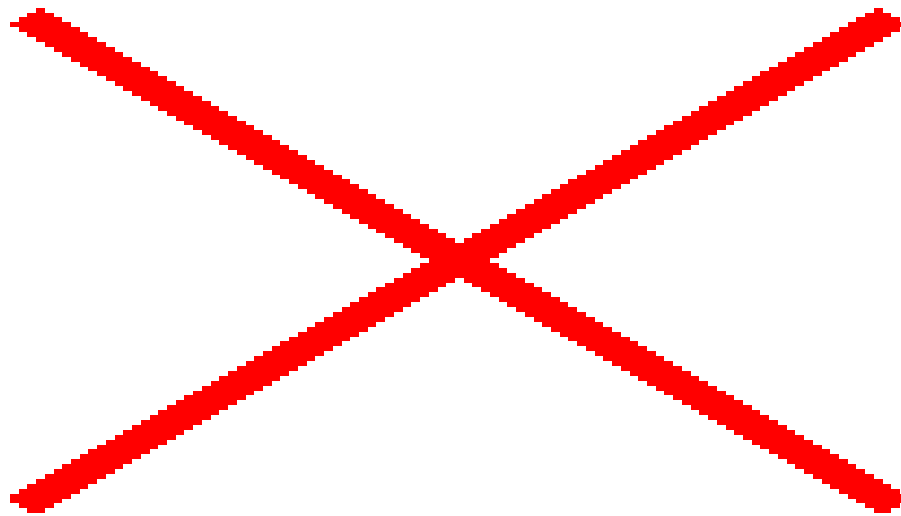
Question 01: Age

Table 15: Age Distribution of Diabetic Patients

Age Group	Count	Percentage
18–39 years	33	33,7%
40–59 years	37	37,8%
60 years and above	28	28,6%
Total	98	100%

Table

15:Age



Distribution of Diabetic Patients

Analysis:

The 40–59 age group is the largest (38%), followed by 18–39 (34%) and 60+ (29%). Unlike Questionnaire 01, patients are relatively younger on average.

Interpretation:

The high proportion of middle-aged diabetic patients (40–59) reflects the typical peak onset of type 2 diabetes. The unexpectedly high proportion of young adults (34%) with T2D suggests early-onset diabetes, possibly driven by obesity, sedentary lifestyles, and poor sleep — reinforcing the study's central hypothesis.

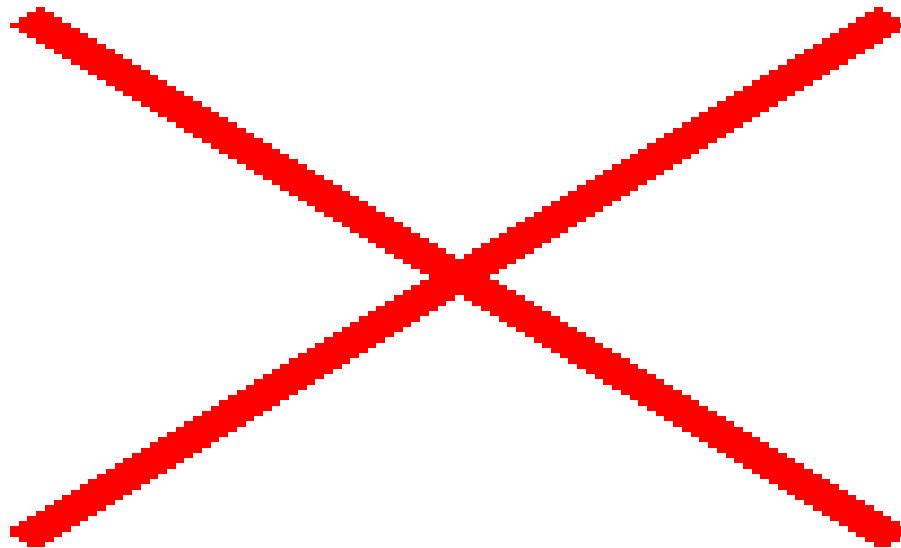
Question 02: Sex

Table 16: Distribution by Sex

ANALYTICAL PHASE

Sex	Count	Percentage
Female	54	55,1%
Male	44	44,9%
Total	98	100%

Table 16: Distribution by Sex



Analysis:

55% of diabetic patients are female and 45% male, a slightly larger female majority than in Questionnaire 01.

Interpretation:

ANALYTICAL PHASE

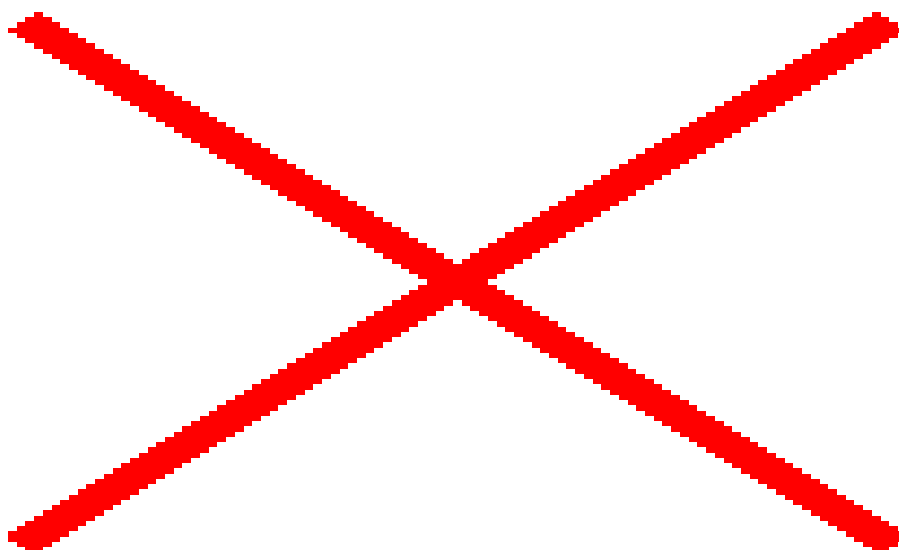
Women are slightly overrepresented in this diabetic sample, consistent with epidemiological data showing women often seek healthcare more frequently. Hormonal factors (e.g., menopause-related metabolic changes) may also contribute to higher T2D prevalence in older women.

Question 03 : Duration of Diabetes

Table 17 : Duration of Diabetes

Duration	Count	Percentage
1 year	42	42,9%
2 years	56	57,1%
Total	98	100%

Table 17:Duration of Diabetes



ANALYTICAL PHASE

Analysis:

57% of patients have had diabetes for 2 years and 43% for 1 year. All patients are recently diagnosed (within 2 years).

Interpretation:

The short disease duration (1–2 years) means patients are in early stages of diabetes management. Sleep quality assessment at this stage is especially valuable as interventions to improve sleep may still significantly influence glycemic trajectory before complications develop.

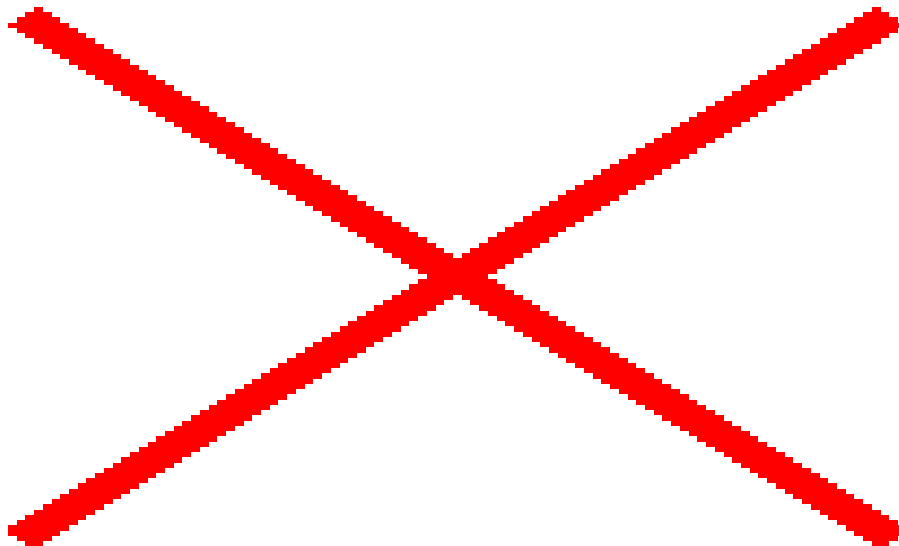
Question 04: Treatment Type

Table 18: Distribution by Treatment Type

Treatment	Count	Percentage
Oral antidiabetics	49	50,0%
Insulin	30	30,6%
Both	19	19,4%
Total	98	100%

Table 18: Distribution by Treatment Type

ANALYTICAL PHASE



Analysis:

50% use oral antidiabetics only, 31% use insulin, and 19% use both. The majority are on first-line oral therapy.

Interpretation:

The relatively high proportion on insulin (31%) and combination therapy (19%) in recently diagnosed patients suggests suboptimal glycemic control, possibly exacerbated by sleep disturbances. Poor sleep is known to impair insulin sensitivity and necessitate higher medication doses.

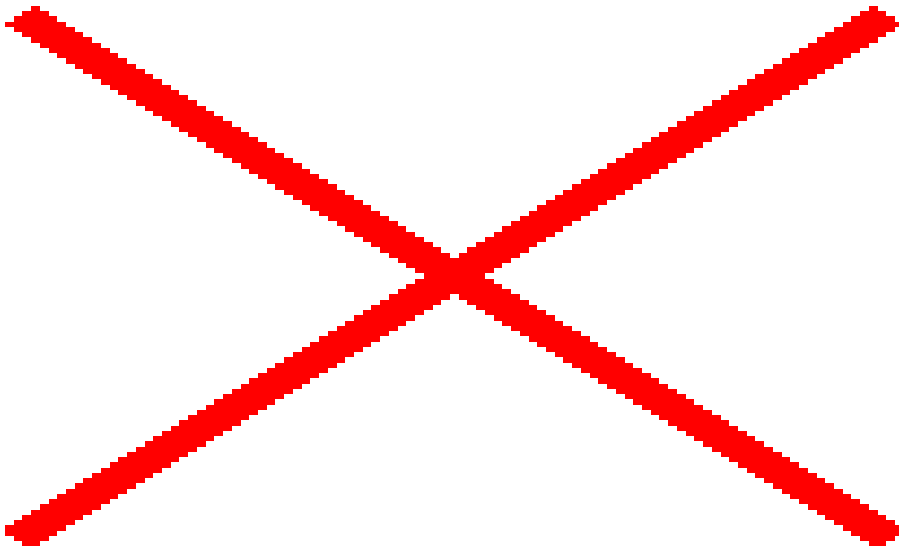
Question 05: HbA1c Level

ANALYTICAL PHASE

Table 19: HbA1c Level Distribution

HbA1c Value	Count	Percentage
<7%	34	34,7%
8%	64	65,3%
Total	98	100%

Table



19:HbA1c Level Distribution

Analysis:

ANALYTICAL PHASE

65% of patients have an HbA1c of 8%, indicating poorly controlled diabetes, while 35% are at 7% (borderline controlled, ADA target $\leq 7\%$).

Interpretation:

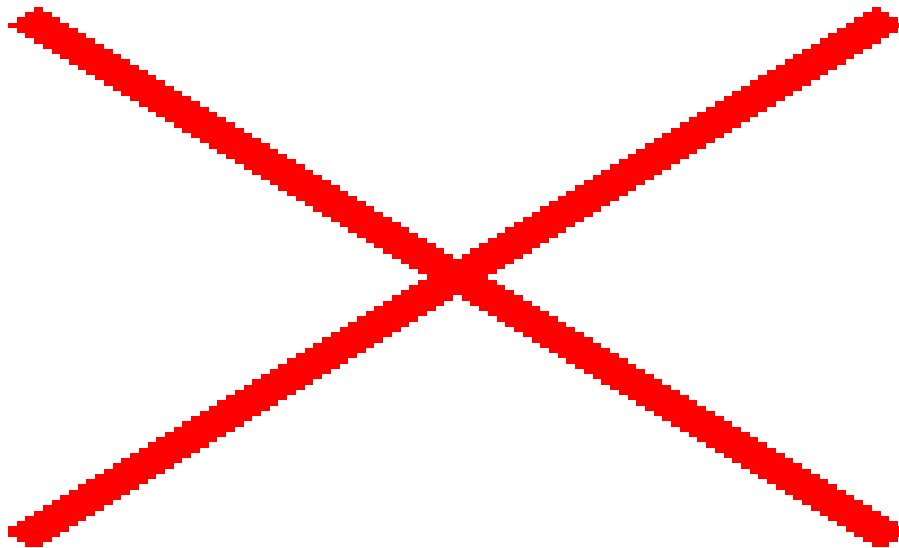
The majority (65%) exceed the ADA-recommended HbA1c target of 7%, confirming inadequate glycemic control. Poor sleep quality is a well-established contributor to elevated HbA1c. These findings strongly support investigating sleep as a modifiable factor in glycemic management.

Question 06: Hours of Sleep per Night

Table 20: Nightly Sleep Duration in Diabetic Patients

Sleep Duration	Count	Percentage
< 5 h	11	11,2%
5–6 h	17	17,3%
6–7 h	23	23,5%
7–8 h	30	30,6%
> 8 h	17	17,3%
Total	98	100%

Table 20: Nightly Sleep Duration in Diabetic Patients



ANALYTICAL PHASE

Analysis:

31% sleep 7–8 h (recommended). However, 28% sleep under 6 hours (11% + 17%), and 17% sleep over 8 hours. Both extremes are clinically concerning.

Interpretation:

Both short sleep (< 6 h) and long sleep (> 8 h) are associated with worse glycemic outcomes in T2D patients. Short sleep increases cortisol and decreases insulin sensitivity, while long sleep may reflect underlying complications or depression — both of which worsen metabolic control.

Question 07: Difficulty Falling Asleep

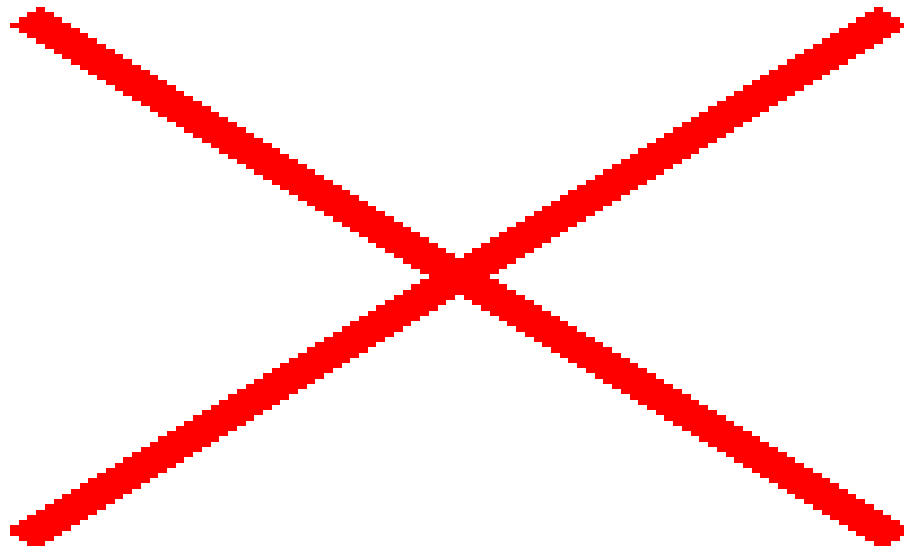
Table 21: Frequency of Difficulty Falling Asleep

Response	Count	Percentage
Never	54	55,1%
Sometimes	44	44,9%
Total	98	100%

Table

21:Frequency of Difficulty Falling Asleep

ANALYTICAL PHASE



Analysis:

45% of diabetic patients sometimes have difficulty falling asleep, compared to 55% who never do.

Interpretation:

Nearly half of patients experience sleep onset difficulties, which can be attributable to nocturia (a known T2D complication), neuropathic pain, anxiety about disease management, or medication side effects — all of which fragment sleep and worsen glycemic control.

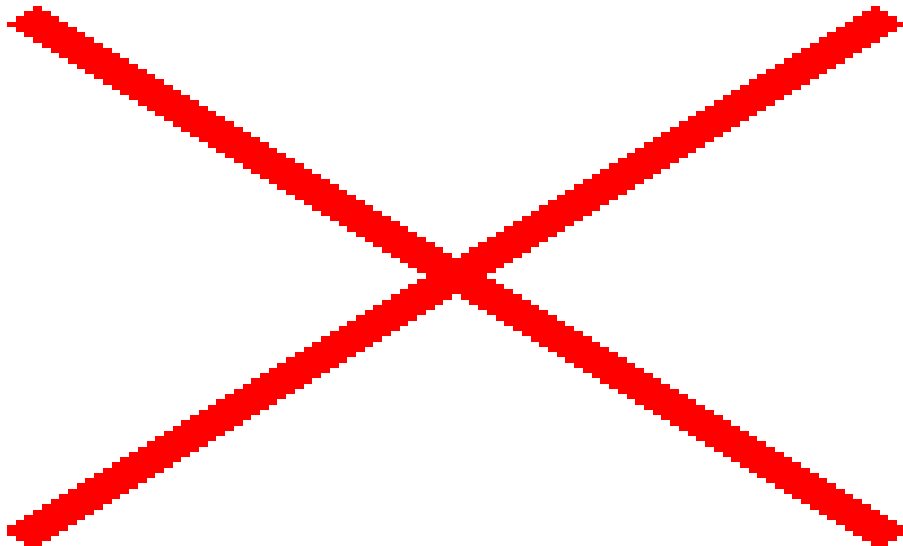
ANALYTICAL PHASE

Question 08: Nighttime Awakenings

Table 22: Nighttime Awakenings in Diabetic Patients

Awakenings	Count	Percentage
None	32	32,7%
1–2	41	41,8%
>2	25	25,5%
Total	98	100%

Table 22:Nighttime Awakenings in Diabetic Patients



Analysis:

ANALYTICAL PHASE

67% of patients experience nighttime awakenings: 42% wake 1–2 times and 26% wake more than twice. Only 33% sleep uninterrupted.

Interpretation:

Frequent awakenings in diabetic patients are often driven by nocturia (excessive nighttime urination), hypoglycemic episodes, or pain from neuropathy. Each awakening interrupts restorative sleep stages, elevating fasting glucose levels and impairing insulin action the following day.

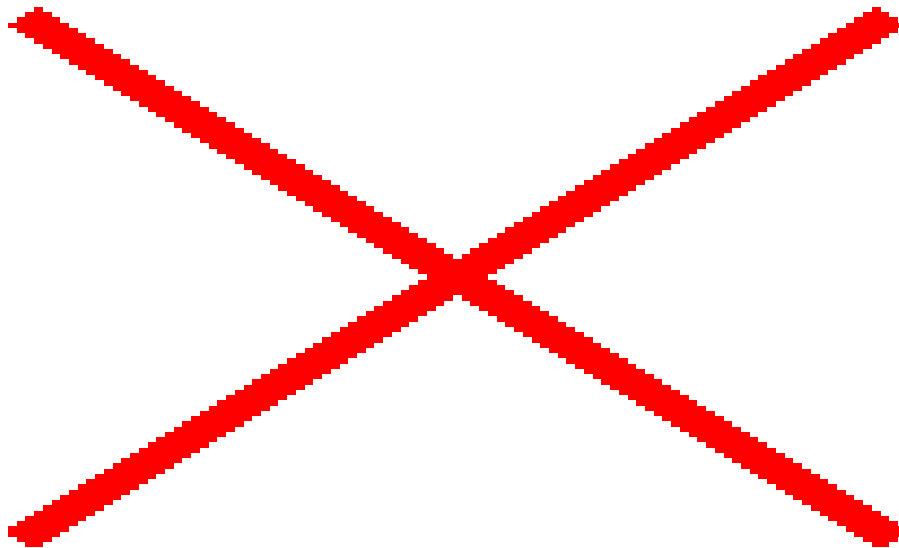
II.5.8 Sleep– Glycaemia Relationship

Question 09: Blood Sugar Higher After Poor Sleep?

Table 23: Self-Reported Blood Sugar Elevation After Poor Sleep

Response	Count	Percentage
Yes	51	52%
No	47	48%
Total	98	100%

Table 23: Self-Reported Blood Sugar Elevation After Poor Sleep



ANALYTICAL PHASE

Analysis:

52% of patients perceive their blood sugar to be higher after a poor night's sleep, while 48% do not notice this effect.

Interpretation:

That over half the patients personally observe a connection between poor sleep and elevated blood sugar provides valuable patient-reported evidence supporting the study's hypothesis. This self-awareness also has clinical implications for patient education and self-management strategies.

Question 10: Increased Hunger After Sleep Deprivation?

Table 24: Self-Reported Hunger Increase After Poor Sleep

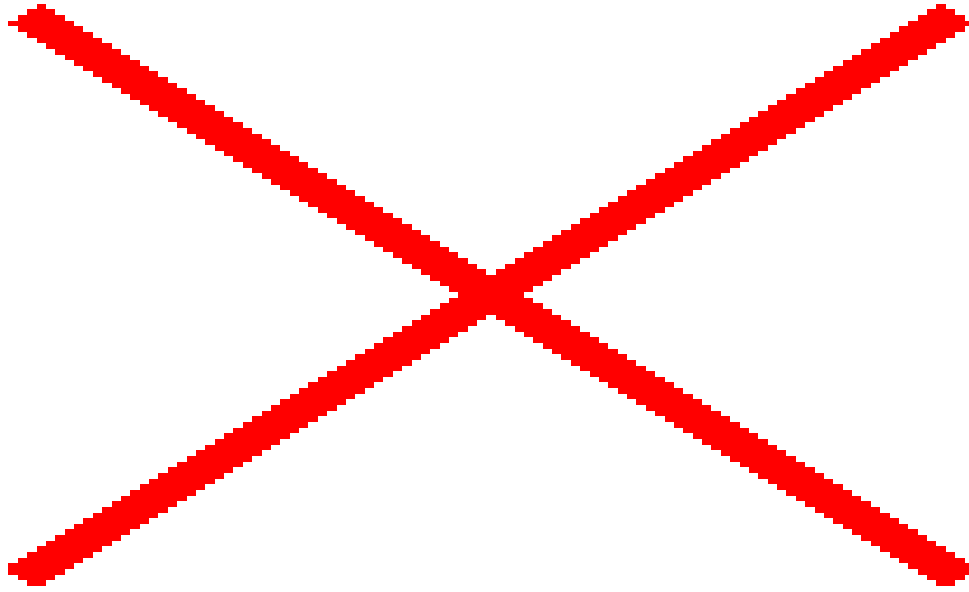
Response	Count	Percentage
Yes	55	56,1%
No	43	43,9%
Total	98	100%

Table

Reported Hunger Increase After Poor Sleep

24:Self-

ANALYTICAL PHASE



Analysis:

56% report increased hunger after sleep deprivation, compared to 44% who do not.

Interpretation:

Increased hunger following sleep loss is driven by elevated ghrelin (hunger hormone) and suppressed leptin (satiety hormone). In diabetic patients, this leads to overconsumption of carbohydrates, directly worsening postprandial glycemia and overall metabolic control.

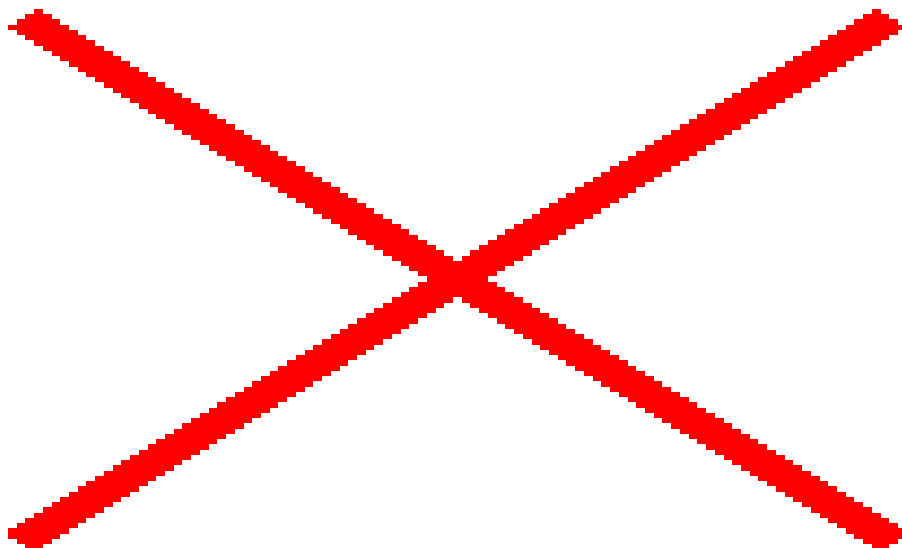
ANALYTICAL PHASE

Question 11: Cause of Nighttime Awakenings

Table 25: Causes of Nighttime Awakenings

Cause	Count	Percentage
Nocturia	31	31,6%
None	25	25,5%
Hypoglycemia	21	21,4%
Pain	21	21,4%
Total	98	100%

Table 25: Causes of Nighttime Awakenings



ANALYTICAL PHASE

Analysis:

Among those with awakenings, nocturia is the most common cause (32%), followed by no identified cause (26%), hypoglycemia (21%), and pain (21%).

Interpretation:

Nocturia as the leading cause of awakening directly reflects poor glycemic control (osmotic diuresis from hyperglycemia). Nocturnal hypoglycemia and neuropathic pain are additional T2D complications impairing sleep, creating a vicious cycle: poor glycemic control → disrupted sleep → worsened glycemic control.

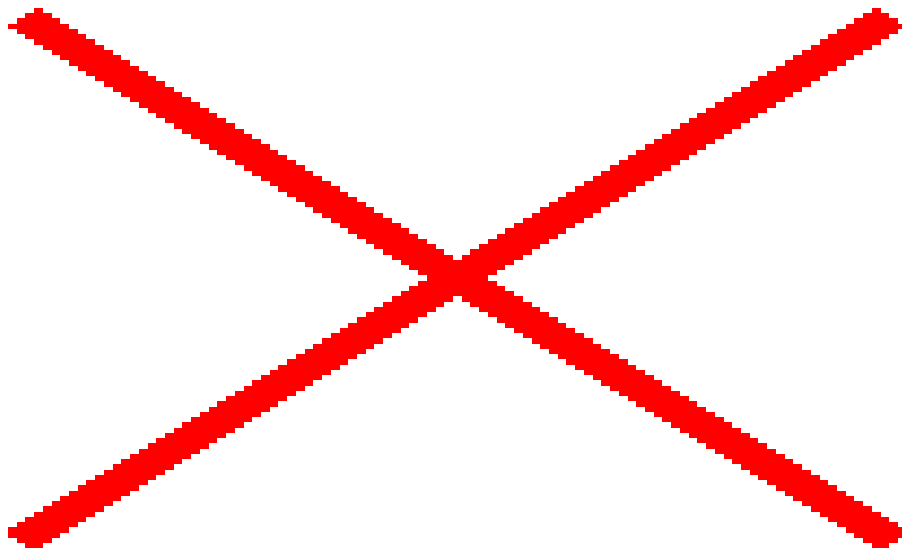
II.5.9 Blood Sugar Monitoring

Question 12: Glycemic Monitoring Frequency

Table 26: Frequency of Blood Glucose Monitoring

Frequency	Count	Percentage
Several times/day	43	43,9%
Once/day	34	34,7%
Rarely or never	21	21,4%
Total	98	100%

Table 26:Frequency of Blood Glucose Monitoring



ANALYTICAL PHASE

Analysis:

44% monitor several times daily, 35% once daily, and 21% rarely or never monitor. 79% conduct regular monitoring.

Interpretation:

While the majority monitor regularly, 21% rarely or never check — a concerning gap given the high HbA1c levels in this population. More frequent monitoring is associated with better glycemic control and may reveal the postprandial and post-sleep glucose spikes described in the literature.

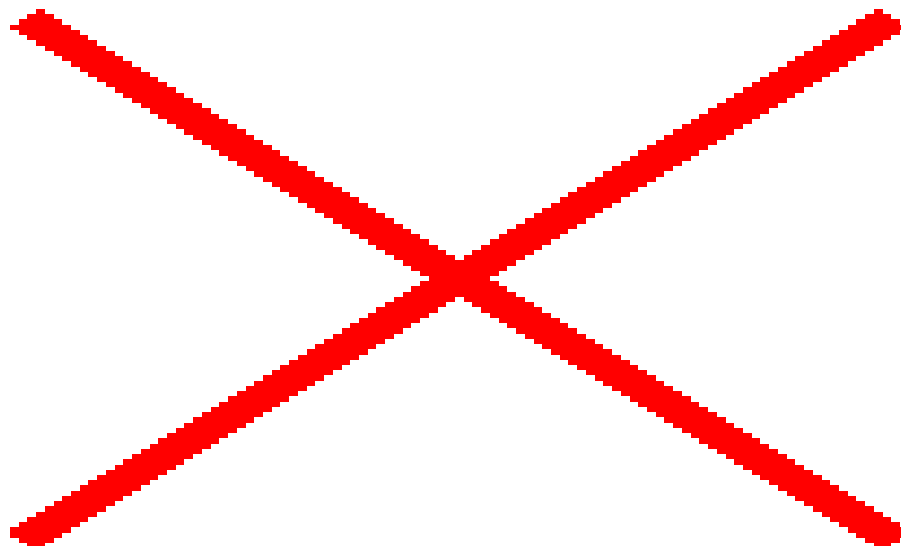
Question 13: Body Weight

Table 27: Body Weight Distribution in Diabetic Patients

Weight	Count	Percentage
65 kg	11	11,2%
70 kg	21	21,4%
85 kg	33	33,7%
95 kg	33	33,7%
Total	98	100%

Table 27: Body Weight Distribution in Diabetic Patients

ANALYTICAL PHASE



Analysis:

68% of patients weigh 85 kg or more (34% at 85 kg and 34% at 95 kg). Only 11% weigh 65 kg and 21% weigh 70 kg.

Interpretation:

The majority of diabetic patients (67.4%) fall within higher body weight categories (85–95 kg). Increased body weight is a well-established risk factor associated with insulin resistance and type 2 diabetes. In combination with other lifestyle factors, such as sleep disturbances, higher body weight may contribute to unfavorable metabolic outcomes

CHAPTER 6: GENERAL DISCUSSION

GENERAL DISCUSSION AND HYPOTHESIS VALIDATION

II.6.1 General Discussion of the Results:

The present study aimed to investigate the relationship between sleep deprivation, insulin resistance, and type 2 diabetes among health-science students and diabetic patients. The results obtained demonstrate that sleep disturbances are highly prevalent in both populations and are associated with several factors known to impair metabolic health.

Among students, a large proportion reported sleeping fewer than seven hours per night, frequent nocturnal awakenings, low levels of physical activity, and regular caffeine consumption. These findings suggest that inadequate sleep is a common behavior in this population and may contribute to metabolic disturbances even before the onset of overt disease. Sleep deprivation has been shown to alter glucose metabolism, reduce insulin sensitivity, and promote hormonal changes that favor weight gain and insulin resistance.

The high prevalence of nighttime awakenings observed among students indicates that sleep quality, in addition to sleep duration, is compromised. Fragmented sleep reduces restorative sleep phases and may interfere with normal endocrine regulation. Consequently, chronic exposure to poor-quality sleep may increase susceptibility to metabolic disorders, particularly among individuals with a genetic predisposition to diabetes.

The results also revealed that many students presented additional risk factors, including family history of diabetes, overweight status, and physical inactivity. The coexistence of these factors with sleep disturbances may create a favorable environment for the development of insulin resistance. Sleep deprivation therefore appears not as an isolated factor but as part of a broader network of behavioral and metabolic determinants.

In diabetic patients, sleep disturbances were equally common. Most participants reported nocturnal awakenings, while a substantial proportion experienced difficulty maintaining continuous sleep. Nocturia was identified as the main cause of sleep interruption. This observation is consistent with the pathophysiology of diabetes, where hyperglycemia promotes increased urine production and consequently disrupts sleep continuity.

Glycemic control was generally unsatisfactory among diabetic patients, as reflected by the predominance of elevated HbA1c values. Furthermore, many participants reported higher blood glucose levels following nights of poor sleep. These findings support the existence of a bidirectional relationship between sleep and diabetes. On one hand, diabetes contributes to sleep disturbances; on the other hand, poor sleep further aggravates metabolic imbalance.

The observation that many diabetic patients reported increased hunger after sleep deprivation is particularly noteworthy. Sleep restriction is known to alter appetite-regulating hormones by decreasing leptin levels and increasing ghrelin secretion. These hormonal changes stimulate food intake, favor weight gain, and may contribute to worsening insulin resistance and glycemic control.

GENERAL DISCUSSION AND HYPOTHESIS VALIDATION

Overall, the findings obtained from both populations demonstrate a consistent relationship between poor sleep quality, sleep deprivation, and metabolic dysfunction. The results support the growing body of evidence suggesting that sleep plays a fundamental role in maintaining glucose homeostasis and metabolic health.

II.6.2 Hypothesis Validation Through Results

Hypothesis 1

A sleep duration of fewer than seven hours per night is associated with an increased risk of glycemic imbalance and insulin resistance.

The results support this hypothesis. A large proportion of students reported insufficient sleep duration, while diabetic patients with poor sleep frequently exhibited suboptimal glycemic control. These observations are consistent with evidence indicating that chronic sleep restriction reduces insulin sensitivity and impairs glucose metabolism.

Hypothesis 2

Poor sleep quality contributes to hormonal and metabolic disturbances involved in insulin resistance.

The frequent occurrence of nocturnal awakenings, difficulty initiating sleep, increased hunger after poor sleep, and self-reported worsening of glycemia supports this hypothesis. These findings are compatible with known physiological mechanisms involving cortisol elevation, leptin reduction, ghrelin stimulation, and impaired insulin action.

Hypothesis 3

Irregular sleep habits are prevalent among university students and may increase future metabolic risk.

The student population demonstrated several unhealthy sleep-related behaviors, including short sleep duration, frequent sleep interruptions, excessive caffeine consumption, and insufficient physical activity. Combined with a high prevalence of family history of diabetes, these factors indicate an increased susceptibility to future insulin resistance.

Hypothesis 4

Sleep deprivation contributes to poorer glycemic control in patients with type 2 diabetes.

Most diabetic participants reported sleep disturbances and elevated HbA1c levels. In addition, many patients perceived worsening blood glucose levels after nights of inadequate sleep. These findings strongly support the existence of a negative impact of sleep deprivation on diabetes management.

GENERAL DISCUSSION AND HYPOTHESIS VALIDATION

II.6.3 Study Limitations

Several limitations should be considered when interpreting the findings:

- The cross-sectional design does not allow the establishment of a causal relationship.
- Sleep quality was assessed using self-reported questionnaires, which may be subject to recall bias.
- Objective sleep measurements such as polysomnography or actigraphy were not available.
- Biological markers directly reflecting insulin resistance were not measured in the student population.
- The sample was limited to a specific geographical region, which may restrict the generalizability of the findings.

Despite these limitations, the study provides valuable information regarding the relationship between sleep disturbances and metabolic health.

GENERAL CONCLUSION AND RECOMMENDATIONS

GENERAL CONCLUSION AND RECOMMENDATIONS

RECOMMENDATIONS:

For Students:

- Promote sleep hygiene education programs within universities.
- Encourage a regular sleep schedule of 7–9 hours per night.
- Reduce evening caffeine consumption.
- Increase physical activity levels.
- Implement periodic screening for metabolic risk factors among students with family histories of diabetes.

For Patients with Type 2 Diabetes :

- Integrate sleep assessment into routine diabetes management.
- Address factors responsible for sleep fragmentation, particularly nocturia.
- Improve glycemic control to reduce diabetes-related sleep disturbances.
- Educate patients regarding the influence of sleep on glucose regulation.

For Healthcare Professionals :

- Consider sleep quality as a modifiable risk factor for insulin resistance and diabetes.
- Include sleep-related questions during routine clinical consultations.
- Develop multidisciplinary approaches integrating sleep medicine and diabetes care.

For Future Research :

- Conduct longitudinal studies to establish causal relationships.
- Use objective sleep assessment methods.
- Investigate hormonal markers such as cortisol, leptin, ghrelin, and HOMA-IR.
- Evaluate the effectiveness of sleep-improvement interventions on glycemic control.

GENERAL CONCLUSION AND RECOMMENDATIONS

GENERAL CONCLUSION:

The present study highlights the important relationship between sleep deprivation, insulin resistance, and type 2 diabetes. The findings reveal a high prevalence of sleep disturbances among both university students and diabetic patients. Short sleep duration, poor sleep quality, frequent nocturnal awakenings, physical inactivity, excess body weight, and family history of diabetes were identified as important factors associated with metabolic risk.

Among diabetic patients, sleep disturbances were associated with poor glycemic control and increased perception of metabolic deterioration following inadequate sleep. These observations support the concept of a bidirectional relationship in which diabetes disrupts sleep and poor sleep further worsens metabolic regulation.

The study confirms that sleep is a fundamental determinant of metabolic health and should be considered alongside nutrition and physical activity in strategies aimed at preventing insulin resistance and type 2 diabetes. Improving sleep quality and duration may represent a simple, low-cost, and effective approach to reducing metabolic risk and enhancing diabetes management.

In conclusion, the results support the central hypothesis of this research: sleep deprivation constitutes an important contributing factor to insulin resistance and plays a significant role in the development and progression of type 2 diabetes.

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APPENDICES

APPENDICES

Appendix A:

Questionnaire 01: University Students – Sleep and Risk of Insulin Resistance

Study Title: Sleep deprivation as a contributing factor to insulin resistance and type 2 diabetes.

All collected information is confidential and used solely for scientific research purposes.

Sociodemographic Data

Age: _____

Sex: ☐ Male ☐ Female

Level of study: ☐ Bachelor's ☐ Master's ☐ Doctorate

Field of study: _____

Family history of diabetes: ☐ Yes ☐ No

Sleep Duration

Average sleep duration per night: ☐ <5 h ☐ 5–6 h ☐ 6–7 h ☐ 7–8 h ☐ >8 h

Bedtime: _____

Wake-up time: _____

Sleep Quality

Difficulty falling asleep: ☐ Never ☐ Sometimes ☐ Often

Nighttime awakenings: ☐ None ☐ 1–2 ☐ >2

Lifestyle

Physical activity: ☐ Sedentary ☐ Moderate ☐ Regular

Caffeine consumption: ☐ None ☐ 1–2/day ☐ >3/day

Anthropometric Measurements

Weight (kg): _____

Metabolic Data

Have you ever had your blood sugar checked? ☐ Yes ☐ No

If yes, please specify the value: _____

APPENDICES

Appendix B:

Questionnaire 02: Sleep Quality and Glycemic Control in Type 2 Diabetic Patients

This questionnaire is intended for patients with type 2 diabetes. Responses will remain strictly confidential.

Clinical Data

Age: _____

Sex: ☐ Male ☐ Female

Duration of diabetes (years): _____

Treatment: ☐ Oral antidiabetics ☐ Insulin ☐ Both

Last HbA1c value (%): _____

Sleep Duration

Number of hours of sleep: ☐ <5 h ☐ 5–6 h ☐ 6–7 h ☐ 7–8 h ☐ >8 h

Sleep Quality

Difficulty falling asleep: ☐ Never ☐ Sometimes

Nighttime awakenings: ☐ None ☐ 1–2 ☐ >2

Relationship Between Sleep and Blood Sugar

Higher blood sugar after a poor night's sleep? ☐ Yes ☐ No

Increased hunger sensation after sleep deprivation? ☐ Yes ☐ No

Cause of nighttime awakenings: ☐ Nocturia ☐ Hypoglycemia ☐ Pain ☐ None

Glycemic Monitoring

☐ Several times a day

☐ Once a day

☐ Rarely or never

Anthropometric Measurements

Weight (kg): _____