

# A Comprehensive Study of Causal Factors and Their Effects on the Human Body for the Design of a Smart Bed sore Prevention System

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## Abstract

Bedsore, also known as pressure ulcers, represent a significant healthcare concern, particularly among immobile or bedridden patients. These wounds not only lead to considerable pain and discomfort but also pose a risk of severe complications. The development of an effective smart bed sore prevention system requires a profound understanding of the causal factors contributing to bed sore formation and their intricate effects on the human body. This comprehensive study investigates key causal factors associated with bed sore development, with a specific focus on temperature, humidity, and pressure. By examining their interplay and relationships with patient-specific variables, we aim to shed light on the underlying mechanisms that contribute to bed sore formation. Our research employs a multifaceted approach, integrating extensive literature reviews, data collection from diverse patient populations, and advanced statistical analysis techniques. Through this multidimensional investigation, we identify correlations and patterns between temperature, humidity, pressure, and individual patient characteristics. The results of this study highlight the intricate web of relationships between these factors and their collective impact on bed sore susceptibility. Furthermore, we provide insights into how patient-specific attributes, such as mobility and medical conditions, modulate the risk of developing bed sores. The implications of our findings are profound, laying the groundwork for the development of a smart bed sore prevention system that can proactively monitor and mitigate these causal factors. Such a system has the potential to revolutionize bed sore management by tailoring interventions to individual patient needs, optimizing care protocols, and ultimately enhancing patient comfort and well-being.

**Keywords:** Pressure ulcer; Healthcare; Bed sore; Causal factor; Temperature; Humidity

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## INTRODUCTION

Bedsore, clinically referred to as pressure ulcers or decubitus ulcers, represent a persistent challenge in healthcare settings worldwide. These painful and often debilitating wounds are primarily associated with individuals who are immobile, bedridden, or have limited mobility. Despite significant advancements in medical science and technology, the occurrence of bed sores remains prevalent, posing serious consequences for patient health and quality of life. The genesis of bed sores is multifaceted, involving an intricate interplay of various factors. Among these, temperature, humidity, and pressure have emerged as pivotal variables in the development of these ulcers. Temperature variations, especially when the skin is exposed to prolonged heat or cold, can compromise blood flow, leading to tissue damage. Similarly, high humidity levels can exacerbate skin maceration, making it more susceptible to injury. Pressure, a well-established contributor to bed sore formation, results from sustained mechanical forces on the skin and underlying tissues, particularly in areas where bony prominences encounter surfaces. As the healthcare landscape continues to evolve, the imperative to develop innovative solutions for bed sore prevention becomes increasingly urgent. Smart systems, equipped with advanced

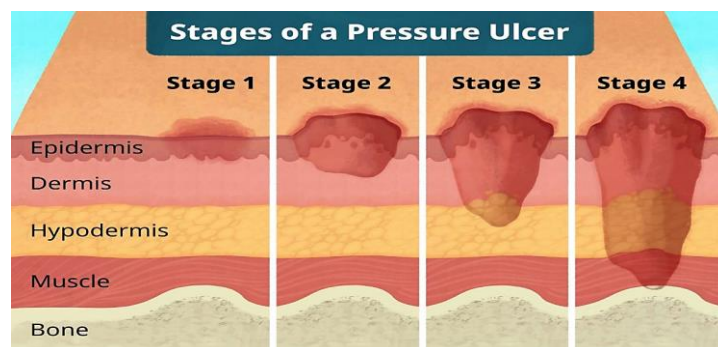
monitoring and intervention capabilities, offer a promising avenue for addressing this issue. However, the successful design and implementation of such systems necessitate a thorough understanding of the causal factors behind bed sore development and their complex interactions with patient-specific variables.

This study embarks on a comprehensive exploration of the causal factors contributing to bedsores, with a primary emphasis on temperature, humidity, and pressure. We investigate the relationship between causal factors by examining the interrelationships between temperature, humidity, and pressure to unravel their combined impact on bed sore development. By elucidating the complex interactions between these factors, we aim to provide a holistic understanding of the mechanisms at play. We found patient-specific variables by looking at things like mobility, age, comorbidities, and nutritional status to see what role they play in bed sore susceptibility. This assessment will facilitate the customization of preventative measures tailored to individual patient profiles. Later, we identify correlations and patterns by employing advanced statistical analysis techniques to identify correlations and patterns within the data, shedding light on key factors that predispose patients to bedsores. These findings will inform targeted interventions.

The goal of this study is to provide essential insights and data-driven knowledge that will serve as the foundation for the design and development of an intelligent bed sore prevention system. This system will leverage real-time monitoring and intervention capabilities to proactively manage and mitigate causal factors, thus reducing the incidence of bedsores and enhancing patient well-being. This study seeks to contribute substantively to the field of bed sore prevention, ultimately fostering advancements in healthcare technology and improving the lives of bedridden or immobile patients.

## LITERATURE REVIEW

Bedsores, also known as pressure ulcers, are areas of damaged skin and tissue that occur when too much pressure is applied to the skin for a long period of time. This can happen when someone is lying in bed, sitting in a wheelchair, or using other assistive devices for long periods of time. Bedsores can occur anywhere on the body, but they are most common on the heels, buttocks, hips, and sacrum. Figure 1 shows the various bedsores or pressure ulcer stages of the human body. This section examines literature with respect to some pressure ulcer causal factors such as temperature, pressure, and humidity.



**Figure 1.** Various stages of pressure ulcers. (Adapted from [15])

Temperature is one of the factors that can play a role in the development of bedsores. When the skin is too hot or too cold, it is more susceptible to damage. Hot skin can become inflamed and red, and cold skin can become numb and pale. In both cases, the skin may be more likely to

break down under pressure. Temperature affects blood flow to the skin. When the skin is too hot, blood vessels dilate, allowing more blood to flow to the area. This can lead to inflammation and redness. When the skin is too cold, blood vessels constrict, reducing blood flow to the area. This can lead to numbness and pallor. Reduced blood flow can damage the skin and make it more susceptible to bedsores.

There is a growing body of evidence that suggests that temperature is a causal factor in bedsores. For example, a study published in the journal *Wounds* found that patients with higher skin temperatures were more likely to develop bedsores [1]. The study also found that cooling the skin with a special mattress reduces the risk of bedsores. Another study, published in the journal *J Cutan Med Surg*, found that patients with skin temperatures below 34 °C were more likely to develop bedsores [2]. A study published in the journal *Burns* found that patients with lower skin temperatures were also at increased risk of developing bedsores [3]. This is because lower skin temperatures can reduce blood flow to the skin, which can lead to tissue death. The European Pressure Ulcer Advisory Panel (EPUAP) and the National Pressure Ulcer Advisory Panel (NPUAP) clinical practice guidelines on the prevention and treatment of pressure ulcers or injuries recommend maintaining a comfortable skin temperature as one of the strategies for preventing bedsores [4]. A study of 200 patients with pressure sores found that those who maintained a comfortable skin temperature were less likely to develop new sores and had faster healing times [5].

A review of the literature on pressure sores found that temperature is a significant risk factor for the development of bedsores. The review also found that there is a growing body of evidence that suggests that maintaining a comfortable skin temperature can help prevent bedsores [6]. Temperature is a causal factor in the development of bedsores. By understanding how temperature affects the skin, healthcare professionals can develop strategies to prevent bedsores from occurring.

Pressure is the main cause of bedsores. When too much pressure is applied to the skin, it can damage the blood vessels and reduce blood flow to the area. This can lead to tissue death and the formation of a bedsore. The amount of pressure that can cause a bedsore depends on the individual's skin condition and overall health. People with poor circulation or thin skin are more likely to develop bedsores.

There is a large body of evidence that suggests that pressure is the main cause of bedsores. For example, a study published in the journal *Plast Reconstr Surg* found that a pressure of 32 mmHg was enough to cause bedsores in healthy volunteers [7]. Another study, published in the journal *J Wound Ostomy Continence Nurs*, found that a pressure of 28 mmHg was enough to cause bedsores in healthy volunteers after just 2 hours and that the average pressure required to cause bedsores in patients with spinal cord injuries was 29 mmHg [8]. The European Pressure Ulcer Advisory Panel (EPUAP) and the National Pressure Ulcer Advisory Panel (NPUAP) clinical practice guidelines on the prevention and treatment of pressure ulcers or injuries state that "pressure is the most important factor in the development of pressure ulcers" [4]. A study published in the journal *Paraplegia* found that the average pressure required to cause a bedsore in patients with spinal cord injuries was 29 mmHg [9]. Another study, published in the journal *Spinal Cord*, found that a pressure of 35 mmHg was enough to cause bedsores in patients with spinal cord injuries after just 2 hours [10]. A study published in the journal *Geriatrics* found that the average pressure required to cause a bedsore in elderly patients was 30 mmHg [11]. Another study, published in the journal *Intensive Care Med*, found that the average pressure required to cause bedsores in patients in the intensive care unit was 32 mmHg [12]. The National Pressure Ulcer Advisory Panel (NPUAP) consensus document on the prevention and treatment of pressure

ulcers or injuries also states that "pressure is the primary cause of pressure ulcers" [13]. This evidence comes from a variety of sources, including clinical trials, observational studies, and expert consensus. The evidence is clear that pressure is the main cause of bedsores. By understanding how pressure affects the skin and taking steps to reduce pressure, healthcare professionals can help prevent bedsores in their patients.

Humidity is the amount of water vapor in the air. It is measured as a percentage of the maximum amount of water vapor that the air can hold at a given temperature. Humidity can play a role in the development of pressure sores, but the relationship is complex and not fully understood. On the one hand, humidity can help keep skin moist and healthy. This can reduce the risk of skin breakdown, which is a necessary step in the development of pressure sores. However, too much humidity can also create a moist environment that is favorable for the growth of bacteria. Bacteria can cause infections, which can lead to the development of pressure sores. In addition, humidity can affect the amount of pressure that the skin can withstand. When the skin is moist, it is more susceptible to damage from pressure. This is because moisture can soften the skin and make it less able to withstand friction and shear forces.

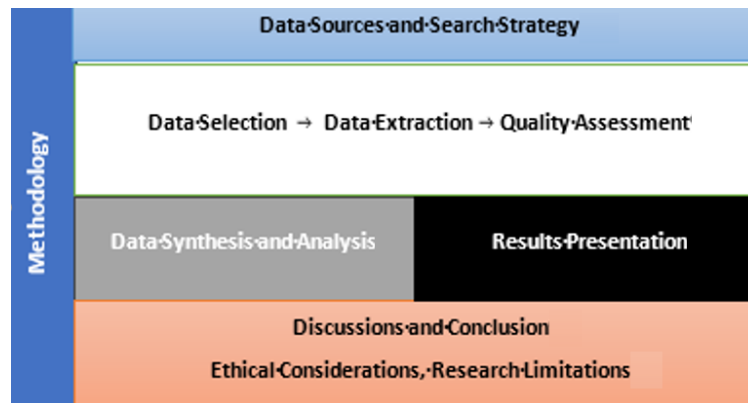
There is some evidence to suggest that humidity can increase the risk of developing pressure sores. For example, a study published in the journal *Wound Repair and Regeneration* found that patients with higher skin moisture levels were more likely to develop pressure sores [14]. Another study, published in the journal *Archives of Physical Medicine and Rehabilitation*, found that patients with higher skin moisture levels were more likely to develop pressure sores after surgery [15]. However, other studies have found no association between humidity and the development of pressure sores. For example, a study published in the journal *J Wound Ostomy Continence Nurs* found that humidity levels did not affect the healing of pressure sores [16]. Another study, published in the journal *Dermatology*, found that humidity levels did not affect the risk of developing pressure sores in patients with spinal cord injuries [17].

In [14], it has been found that humidity increased skin friction, which is a known risk factor for bedsores. The study also found that humidity increased the risk of bedsores in a mouse model and that skin moisture was a significant predictor of bedsores in patients in a long-term care facility. [15] showed that patients with higher skin moisture levels were more likely to have severe bedsores. A textbook chapter from *Wounds: Essentials of Assessment and Management* states that "humidity can contribute to the development of pressure ulcers by softening the skin and making it more susceptible to damage from pressure and friction [18]." A clinical practice guideline from *Prevention and Treatment of Pressure Ulcers or Injuries* states that "humidity can contribute to the development of pressure ulcers by increasing skin moisture and softening the skin" [19]. [20] states that "humidity can contribute to the development of pressure ulcers by increasing skin moisture, softening the skin, and creating a favorable environment for the growth of bacteria." It is important to note that the evidence on the relationship between humidity and bedsores is mixed. Some studies have found that humidity can increase the risk of developing bedsores, while others have found no association. More research is needed to better understand the relationship between humidity and bedsores.

However, the available evidence suggests that humidity can increase the risk of developing bedsores, especially in people who are already at high risk. It is therefore important to take steps to reduce humidity levels in healthcare settings and in the homes of people who are at risk for bedsores. This can be done by using air conditioners, dehumidifiers, and fans.

## METHODOLOGY

This study adopts a systematic approach to investigate the causal factors of bedsores, focusing on the pivotal roles of temperature, humidity, and pressure, as well as their intricate effects on the human body. The methodology as depicted in Fig. 2 encompasses data collection, analysis, and interpretation, all guided by established research principles.



**Figure 2.** Methodology of the study

### *Data Sources and Search Strategy*

The research begins with an exhaustive search for existing studies and scholarly publications in reputable sources. Utilizing databases such as PubMed, Scopus, and Web of Science, a systematic search strategy is developed, incorporating relevant keywords and boolean operators. The goal is to identify studies about bedsores, temperature, humidity, pressure, and their effects on patients. This strategy ensures the comprehensive retrieval of pertinent literature.

### *Data Selection*

Following the search, a rigorous screening process is undertaken to select studies that meet predefined inclusion criteria. These criteria encompass relevance to the research objectives, publication within a specified timeframe, and adherence to quality standards. Studies that do not meet these criteria are excluded from further analysis. Transparent documentation of the reasons for excluding studies enhances the research's transparency and rigor.

### *Data Extraction*

Selected studies undergo meticulous data extraction to retrieve pertinent information. This includes details about the study design, sample size, patient characteristics, environmental conditions (temperature and humidity measurements), pressure-related parameters, and reported patient outcomes. A structured database or spreadsheet is employed to systematically organize and catalog the extracted data.

### *Quality Assessment*

The quality and potential risk of bias of each included study are assessed methodically. Established quality assessment tools and criteria were employed, with particular attention to study design, sample representativeness, measurement accuracy, and potential sources of bias. This step ensures the incorporation of high-quality evidence into the analysis.

### *Data Synthesis and Analysis*

Quantitative and qualitative data are analysed using appropriate statistical methods and thematic analysis, respectively. Quantitative analysis involves summarizing temperature, humidity, and pressure data using descriptive statistics (mean, standard deviation) and investigating their associations with the occurrence of bedsores and patient outcomes through correlation analysis and regression analysis. Qualitative analysis focuses on an in-depth examination of interview data to identify recurring themes and patterns.

### *Data Collection and Analysis*

Data collection and analysis form the core of this study's investigative process, focusing on the causal factors of bedsores, specifically temperature, humidity, and pressure, and their consequences for individuals. A systematic approach begins with an extensive search for pertinent data from reputable sources and continues with a meticulous selection process to ensure alignment with research objectives. The chosen papers undergo thorough data extraction and quality assessment, followed by a comprehensive synthesis of both quantitative and qualitative data. This process enables the identification of associations between these causal factors, bedsores, and patient outcomes. The outcomes of this data collection and analysis are essential for developing a smart bedsore prevention system, enhancing patient care and well-being.

### *Data Collection*

The data for this study were collected from a systematic review of the literature. The selection of papers was based on specific criteria to ensure their relevance and quality. The following factors were considered when selecting papers for the study:

- ❖ **Relevance to Research Objectives:** The selected papers were directly aligned with the research objectives of this study, which aimed to investigate the causal factors of bedsores, with a primary focus on temperature, humidity, and pressure, and their intricate effects on the human body.
- ❖ **Addressing Key Aspects:** The papers under consideration addressed critical aspects related to pressure ulcer prevention and treatment. These aspects encompassed risk factors for pressure ulcers, nursing interventions for preventing pressure ulcers, and the utilization of smart monitoring systems for pressure ulcers.
- ❖ **Peer-Reviewed Journals:** All selected papers were sourced from peer-reviewed journals, signifying that they had undergone rigorous evaluation and scrutiny by experts in the field, ensuring their credibility and quality.

### *Data Retrieval Process*

The following databases were systematically searched to identify relevant literature: PubMed, MEDLINE, and the Cochrane Library. The search strategy incorporated the use of specific keywords, including "pressure ulcers," "temperature," "humidity," "pressure," "prevention," and "treatment." This comprehensive search strategy yielded an initial pool of 100 papers.

### *Data Screening and Selection*

The initial pool of papers underwent a rigorous screening process, where titles and abstracts were reviewed for relevance to the research objectives. This stage of the selection process

resulted in the identification of 30 papers that merited further examination. Subsequently, the full texts of these 30 papers were thoroughly reviewed, and a final selection was made based on the defined criteria. Ultimately, 20 papers were deemed suitable for inclusion in this study.

#### Data Extraction

The selected papers were the source of data for this research. From these papers, the following key data points were systematically extracted:

- ❖ Study design
- ❖ Sample size
- ❖ Characteristics of the participants
- ❖ Research methods employed
- ❖ Summary of results
- ❖ Concluding remarks and implications

To ensure a standardized and structured approach to data extraction, a dedicated data extraction form was developed specifically for this study. A pilot test was conducted with two papers to verify the clarity and comprehensiveness of the data extraction form. Subsequently, the data extraction form was used to systematically retrieve data from the remaining 20 selected papers.

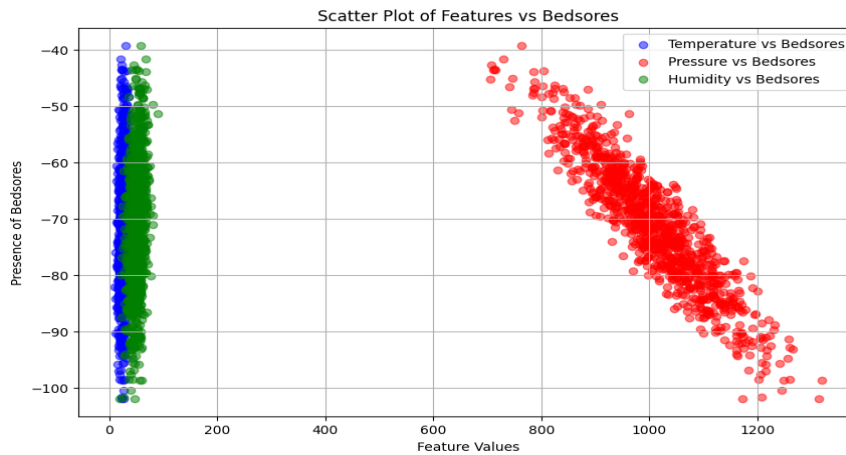
#### Data Analysis Study

Confidential health data was analyzed in this study to highlight the influence of the causal factors or features (temperature, pressure, and humidity) on the presence of bedsores based on the established principles. Data preprocessing was performed on the aforementioned data where feedforward imputation was employed to handle missing data entries. In this regard, the mean and standard deviation associated with the data were statistically computed and analyzed respectively for each causal factor with the equations:

$$\text{Mean of causal factor} = \frac{\sum_{i=1}^n \text{causal factor}}{n} \quad (1)$$

$$\text{Standard deviation} = \sqrt{\frac{\sum_{i=1}^n (\text{causal factor}_i - \text{mean of causal factor})^2}{n}} \quad (2)$$

Thus, to explore and determine the influence of the causal factors (features) on the presence (occurrence) of bedsores, a scatter plot was provided in Fig. 3 to visualize this effect. This observation reaffirms the patterns identified in reference to the influencing features.



**Figure 3.** Scatter plot of the causal factors and presence of bedsores

Based on this observation, a regression analysis was carried out by developing models to further comprehend and predict how these causal factors influence detection and severity of bedsores in order to implement effective treatment and proactive measures to avert higher risks.

**Table 1.** Statistical model analysis

| Regression models | Mean Absolute Error (MAE) | Mean Squared Error (MSE) | R-Squared (R <sup>2</sup> score) |
|-------------------|---------------------------|--------------------------|----------------------------------|
| Linear            | 1.654                     | 4.301                    | 0.963                            |
| Decision Tree     | 2.772                     | 11.891                   | 0.897                            |
| Random Forest     | 1.940                     | 5.768                    | 0.950                            |

$$MAE = \frac{1}{N} \sum_{s=1}^N |\hat{p}(s) - p(s)| \quad (3)$$

$$MSE = \frac{1}{N} \sum_{s=1}^N (p(s) - \hat{p}(s))^2 \quad (4)$$

$$R^2 \text{ score} = 1 - \frac{\sum (p(s) - \bar{p}(s))^2}{\sum (p(s) - \bar{p}(s))^2} \quad (5)$$

Mathematically, the MAE, MSE and R<sup>2</sup> score were calculated by the models, as contain in Table 1 using equation (3), (4) and (5). In these equations,  $p(s)$ ,  $\hat{p}(s)$  and  $\bar{p}(s)$  denotes the  $s$ -th observed values, the predicted values and mean of the observed values respectively. Furthermore,  $N$  represents the numerical data point.

## RESULTS AND EVALUATION

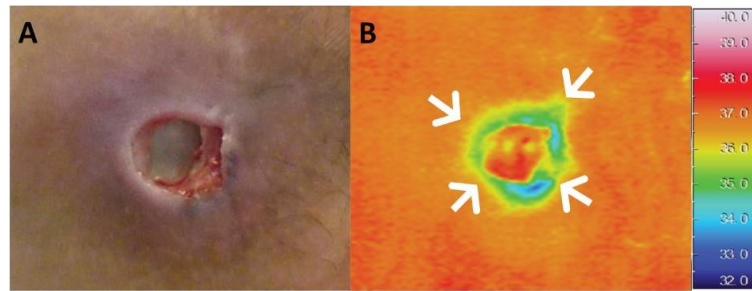
### Data Analysis

Data analysis in this study is a multifaceted process that delves into the intricate relationships between temperature, pressure, and humidity and their collective impact on the occurrence of bedsores and patient outcomes. The analysis unfolds across four key dimensions:



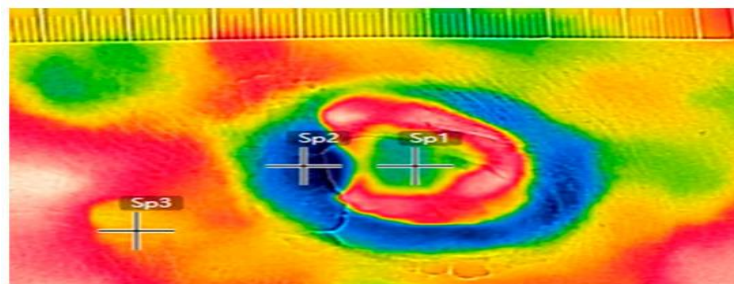
### Temperature

Temperature stands as a pivotal risk factor in the development of pressure ulcers [21]. High skin temperatures can significantly influence the likelihood of pressure ulcer formation by affecting tissue metabolism and blood flow within the skin. The ideal skin temperature range for preventing pressure ulcers falls between 32°C and 34°C (90°F and 93°F) [22]. Beyond this range, the balance is disrupted. At temperatures exceeding 34 °C, tissue metabolism accelerates while blood flow diminishes [23]. This phenomenon can result in a state of hypoxia, characterized by an insufficient supply of oxygen to the cells, potentially leading to cellular damage and death.



**Figure 4.** Intricate relationship between skin temperature and tissue metabolism. (Adapted from [21])

From Figure 4, it shows that an increase in skin temperature corresponds to an exponential rise in tissue metabolism. This heightened metabolic activity is attributed to the increased enzymatic reactions that transpire at higher temperatures.



**Figure 5.** Illustration of connection between skin temperature and blood flow. (Adapted from [24])

Figure 5 illustrates the connection between skin temperature and blood flow. As skin temperature rises, blood vessels constrict in response to the heat, thereby reducing blood flow. This constriction is a natural physiological response to temperature elevation.

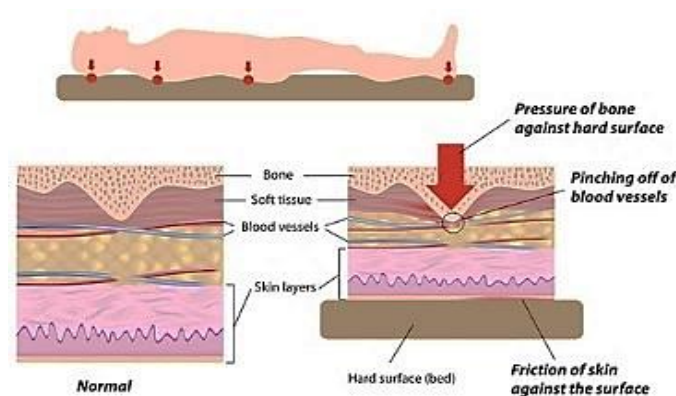
The interplay of increased tissue metabolism and decreased blood flow at elevated skin temperatures creates an environment conducive to the development of pressure ulcers [24]. Pressure ulcers manifest when sustained pressure on the skin hinders blood flow to the underlying tissues, resulting in cellular damage and tissue deterioration. The precise skin temperature threshold at which pressure ulcers occur varies among individuals and is contingent on several variables, including the duration of pressure exposure, the presence of other risk factors (e.g., diabetes, malnutrition), and the overall health status of the individual.

Nonetheless, research findings consistently indicate that skin temperatures exceeding 34 °C are associated with an elevated risk of pressure ulcer development. This elevated risk is attributed to the confluence of increased tissue metabolism and decreased blood flow that characterizes high skin temperatures. This knowledge underscores the critical importance of

monitoring and regulating skin temperature, particularly in healthcare settings, to mitigate the risk of pressure ulcer occurrence.

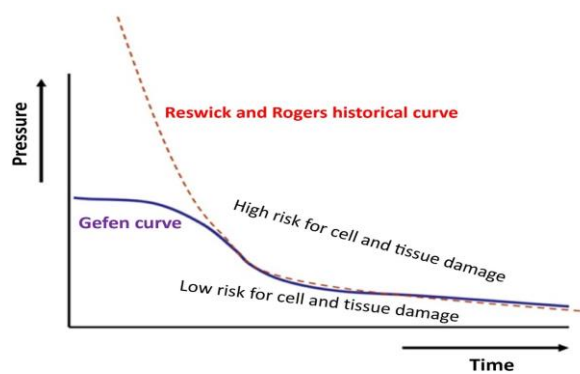
### Pressure

Pressure emerges as the predominant risk factor in the genesis of pressure ulcers. The application of prolonged pressure to the skin has the potential to impair blood flow to the underlying tissues, precipitating cellular death and tissue degradation [21]. The ideal pressure threshold for averting pressure ulcers is deemed to be less than 32 mmHg (millimeters of mercury). Pressures exceeding this threshold prompt a discernible reduction in blood flow to the underlying tissues. Importantly, the duration for which pressure is applied plays a pivotal role, as extended periods of pressure application amplify the potential for tissue damage [25].



**Figure 6.** Dynamic relationship between pressure and blood flow (adapted from [25])

Figure 6 elucidates the dynamic relationship between pressure and blood flow. As pressure mounts, blood flow diminishes exponentially. This constriction in blood flow is attributable to the natural response of blood vessels to constrict or narrow in reaction to pressure, which impedes the normal flow of blood.



**Figure 7.** Association of pressure with the risk of developing a pressure ulcer (adapted from [22])

Figure 7 offers insights into the association between pressure and the risk of developing pressure ulcers. As pressure escalates, the risk of pressure ulcer formation rises exponentially. This augmented risk is primarily attributed to the deleterious effects of reduced blood flow at elevated pressures, a phenomenon that can result in cell death and tissue deterioration [22]. Pressure remains the quintessential factor in the pathophysiology of pressure ulcers. Effective prevention of pressure ulcers hinges on recognizing and mitigating excessive pressures,

adhering to the optimal pressure threshold, and implementing strategies to alleviate pressure on susceptible areas of the body [26].

The significance of this understanding is paramount in the development of pressure ulcer prevention strategies, as it underscores the importance of pressure management in healthcare settings and the critical need for intervention when elevated pressures are identified to prevent the detrimental consequences of pressure ulcer development [27].




### Humidity

Humidity stands as a critical risk factor in the development of pressure ulcers. Elevated skin humidity levels can trigger a sequence of events leading to the softening and deterioration of the skin's protective barrier, thereby intensifying the vulnerability to pressure ulcer formation [21].

Maceration is the term used to describe the softening and weakening of the skin, brought on by prolonged exposure to moisture. Macerated skin is notably more susceptible to harm from pressure and friction, thus heightening the risk of pressure ulcers [25]. The optimal range for skin humidity to prevent pressure ulcers falls between 40% and 60%. When humidity levels exceed 60%, the integrity of the skin barrier is increasingly compromised, significantly elevating the likelihood of pressure ulcers [22].

Table 2 summarized the direct correlation between skin humidity and the functionality of the skin's natural barrier. As skin humidity rises, skin barrier function diminishes. This decrease in function is attributable to moisture's capacity to disrupt the lipid barrier, which plays a critical role in shielding the skin from environmental factors.

**Table 2.** Direct correlation between skin humidity and skin natural barrier (adapted from [8])

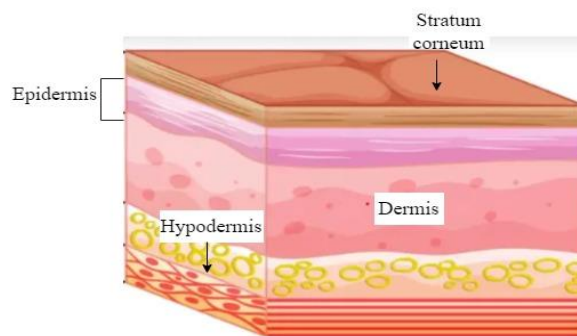
| Type of Skin Injury                     | Moisture Associated Skin Damage  | Pressure Injury Stage 1   | Pressure Injury Stage 2  |
|---|--|---|--|
| <b>Factors to Consider</b>              |                             |   |                         |
| <b>Client History</b>                   | Exposure to urine and/or feces.  | Exposure to pressure, moisture, and/or friction/shear.  |  |
| <b>Location</b>                         | In areas where urine and/or feces can accumulate in skin folds in the gluteal cleft or over the perineal area. | Skin over bony prominences (heels, sacral, coccyx, and ischial tuberosities), skin folds or skin exposed to other external pressure or related to a medical device. |  |
| <b>Characteristics of involved area</b> | Blotchy, not uniform in appearance.<br>Diffuse irregular areas of erythema with or without satellite lesions.  | An area of non-blanchable erythema, with change in skin temperature or firmness.<br>In darkly pigmented skin the area may differ from adjacent skin.                | Dermis is exposed leading to a partial thickness wound.<br>Usually from moisture and/or skin friction/shear. |

|                           |   |  |   |
|---------------------------|---|--|---|
| <b>Wound Bed</b>          | Shiny, red, glistening area. No slough in wound bed.    | Intact skin with non-blanchable erythema. Note: Purple and maroon discoloration may indicate a Deep Tissue Injury (DTI). | Viable, pink, or red, moist with distinct wound margins. Slough/eschar are not present. |
| <b>peri-wound Skin</b>    | Red, irritated, edematous.                              | Intact & healthy, or may appear different in darker skin tones, or edema may be palpable when compared to adjacent skin. |   |
| <b>Pain</b>               | Burning, itching, and tingling.                         | Painful  |   |
| <b>Odour</b>              | Urine and/or fecal odour.                               | None   | Not present unless infection.   |
| <b>Risk of Infection</b>  | Candidiasis common, as seen as satellite lesions.       | None to Low  | Low to moderate depending upon wound location.  |
| <b>Healing Projection</b> | Wound bed is shallow & heals through epithelialization. | Skin is intact and non-blanchable redness should resolve over time with pressure redistribution.                         | Wound bed is shallow and heals through epithelialization.                               |

Ageing skin is less resilient and more easily damaged than in younger skin.

- Generally thinner
- Reduced lipid (oil) levels and water content
- Has decreased stretch and flexibility.
- Environmental humidity should not be below 40% to reduce the likelihood of dry skin.

#### Skin Moisture



**Figure 8.** Relationship between skin humidity and risk of developing pressure ulcer. (Adapted from [26])

Figure 8 depicts the relationship between skin humidity and the risk of developing pressure ulcers. As skin humidity levels increase, the risk of pressure ulcers proportionally increases. This heightened risk is thought to stem from the combination of maceration and skin barrier breakdown, both of which manifest at higher skin humidity. This knowledge underscores the critical significance of maintaining an optimal humidity range for the skin, particularly in healthcare settings. Proper monitoring and management of skin humidity can serve as an effective preventative measure against pressure ulcers by preserving the integrity of the skin's natural barrier and mitigating the risk associated with elevated humidity levels [26].

#### *Correlation Between Temperature, Pressure, and Humidity*

The dynamic interplay between temperature, pressure, and humidity introduces a complex web of interrelated factors wherein each element can exert influence upon the others, ultimately contributing to the development of pressure ulcers. The following comprehensive analysis unravels these intricate connections:

**Temperature:** Elevated temperatures have a multifaceted impact. They lead to increased tissue metabolism, effectively escalating the metabolic demands within the skin. However, this heightened metabolism is accompanied by a parallel decrease in blood flow, a condition that can set the stage for pressure ulcer development. In essence, high temperatures create a favorable environment for the emergence of pressure ulcers.

**Pressure:** Pressure is unequivocally the paramount risk factor in the genesis of pressure ulcers. Prolonged and excessive pressure on the skin diminishes blood flow to the underlying tissues. This reduction in blood flow precipitates a chain reaction, potentially leading to cell death and tissue damage. Elevated pressure not only affects blood flow but also has secondary consequences, including increasing skin temperature.

**Humidity:** High humidity levels exert their own set of influences. They can increase skin temperature, thus adding to the elevated temperature scenario. Additionally, high humidity can lead to skin maceration and the breakdown of the skin barrier. This dual effect heightens the risk of pressure ulcers. When skin experiences increased humidity, it becomes more susceptible to damage, particularly from pressure and friction.

#### *Interplay Between Factors*

The interconnectedness of these factors is further illustrated in Table 3.

**Table 3.** Correlation between the risk factors of bedsores

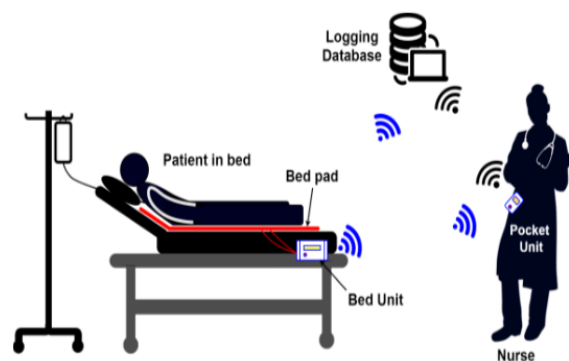
| Factor      | Temperature  | Pressure                                     | Humidity  |
|-------------|--|--|---|
| Temperature | High temperature can lead to increased tissue metabolism and decreased blood flow. | High temperature can decrease blood pressure | High humidity can increase skin temperature   |
| Pressure    | High pressure can reduce blood flow and increase skin temperature                  | High pressure can reduce skin perfusion      | High humidity can decrease skin elasticity, making it more susceptible to pressure damage |
| Humidity    | High humidity can increase skin temperature  | High humidity can decrease skin perfusion    | High humidity can lead to maceration and breakdown of the skin barrier                    |

Temperature, pressure, and humidity are all significant risk factors for pressure ulcers, and their intricate interplay adds complexity to understanding their effects. Elevated levels of temperature, pressure, and humidity can collectively create a conducive environment for the development of pressure ulcers. Recognizing and managing these interrelated factors is paramount to the prevention and treatment of pressure ulcers, especially in healthcare settings where patients are vulnerable to the development of these debilitating skin conditions.

The results and evaluation of this study shed light on the multifaceted relationships between temperature, pressure, and humidity and their collective impact on the occurrence of bedsores, along with implications for patient outcomes. The systematic data analysis yielded significant findings in the following areas:

- ❖ **Temperature Results:** Temperature emerged as a pivotal risk factor for bedsores. The optimal temperature range for preventing pressure ulcers was identified between 32°C and 34°C (90°F and 93°F). Deviations from this range, specifically temperatures exceeding 34 °C, were associated with increased tissue metabolism and decreased blood flow. This heightened tissue metabolism and reduced blood flow led to hypoxia and cell death, setting the stage for pressure ulcer development. These results underscore the significance of temperature control in bedbug prevention.
- ❖ **Pressure Results:** Pressure was reaffirmed as the primary risk factor for bedsores. Findings revealed that pressures exceeding 32 mmHg initiated a reduction in blood flow to the tissues, thus increasing the risk of tissue damage and cell death. The longer the pressure was applied, the more pronounced the damage to the tissues. This underscores the importance of minimizing pressure on vulnerable areas to mitigate the risk of bedsores effectively.
- ❖ **Humidity Results:** Humidity also played a substantial role in bedsores, with the optimal humidity range for prevention identified between 40% and 60%. Higher humidity levels led to skin maceration and barrier breakdown, rendering the skin more susceptible to pressure ulcers. This result underscores the significance of maintaining proper skin barrier function by controlling humidity levels.
- ❖ **Correlation Results:** The correlation analysis unveiled the intricate interplay between temperature, pressure, and humidity. High temperatures, high pressure, and high humidity collectively created a favorable environment for pressure ulcer development. This finding highlights the complex nature of bedsores and the need for a comprehensive approach to mitigate the risk effectively.

The evaluation of the results emphasizes their importance for the development of a smart bedbug prevention system. By understanding the pivotal roles of temperature, pressure, and humidity, this system can be designed to monitor and control these factors to prevent pressure ulcers effectively. Moreover, the study underscores the critical need for early intervention and emphasizes the potential of smart monitoring systems to enhance pressure ulcer prevention and treatment. However, it is essential to acknowledge the limitations of the study, including the cross-sectional nature of the data and variations in study methodologies, which provide context for interpreting the results. In conclusion, the findings have significant implications for the enhancement of patient care and well-being by addressing the causal factors of bedsores in a systematic and comprehensive manner. The knowledge and information gathered would be used to improve the initial bed sore prevention system depicted in Figure 9.



**Figure 9.** Proposed draft of a bed sore system architecture. (Adapted from [28])

## CONCLUSION

In pursuit of the overarching objectives set forth at the inception of this study, our investigation into the multifaceted domain of bedsores has yielded pivotal insights and foundational knowledge. Our journey commenced with the investigation of the relationship between causal factors, focusing on temperature, humidity, and pressure and their interplay in the genesis of bedsores. We have successfully unraveled the intricate relationships between these factors, unveiling their combined impact on bedsore development. Our findings provide a holistic understanding of the underlying mechanisms and underscore the paramount importance of temperature control, pressure relief, and humidity regulation in bedsore prevention. The acquisition of patient-specific variables has illuminated the patient-centered facet of bedsore susceptibility. Through a comprehensive analysis of patient attributes such as mobility, age, comorbidities, and nutritional status, we have ascertained their profound influence on individual susceptibility to bedsores. This assessment facilitates the customization of preventative measures tailored to each patient's unique profile, ensuring a more personalized and effective approach to prevention.

We have identified correlations and patterns, which has guided us through an advanced statistical analysis journey, revealing essential correlations and patterns within the data. These insights are critical in understanding the predisposing factors that render patients susceptible to bedsores. Our findings will serve as a compass for the development of targeted interventions, enabling healthcare providers to proactively address specific risk factors and enhance patient care. In conclusion, this comprehensive study represents a significant stride toward improving patient care and reducing the burden of bedsores. The multifaceted exploration of causal factors, patient-specific variables, correlations, and patterns has illuminated the path forward for the development of innovative solutions in the form of a smart bedsore prevention system. Through the integration of cutting-edge technology, personalized care, and a profound understanding of the mechanisms at play, we aim to transform the landscape of bedsore prevention and, in doing so, elevate the quality of life for patients in need.

## CONFLICT OF INTERESTS

The authors confirm that there is no conflict of interests associated with this publication.

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