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Published in:
Journal of Advanced Nursing

DOI:
10.1111/jan.12405

Publication date:
2014

Citation for published version (Harvard):

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DISCUSSION PAPER

A new pressure ulcer conceptual framework

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Accepted for publication 1 March 2014

Abstract

Aim. This paper discusses the critical determinants of pressure ulcer development and proposes a new pressure ulcer conceptual framework.

Background. Recent work to develop and validate a new evidence-based pressure ulcer risk assessment framework was undertaken. This formed part of a Pressure Ulcer Programme Of reSeArch (RP-PG-0407-10056), funded by the National Institute for Health Research. The foundation for the risk assessment component incorporated a systematic review and a consensus study that highlighted the need to propose a new conceptual framework.


Data Sources. The new conceptual framework links evidence from biomechanical, physiological and epidemiological evidence, through use of data from a systematic review (search conducted March 2010), a consensus study (conducted December 2010–2011) and an international expert group meeting (conducted December 2011).

Implications for Nursing. A new pressure ulcer conceptual framework incorporating key physiological and biomechanical components and their impact on internal strains, stresses and damage thresholds is proposed. Direct and key indirect causal factors suggested in a theoretical causal pathway are mapped to the physiological and biomechanical components of the framework. The new proposed conceptual framework provides the basis for understanding the critical determinants of pressure ulcer development and has the potential to influence risk assessment guidance and practice. It could also be used to underpin future research to explore the role of individual risk factors conceptually and operationally.

continued on page 2223
**Conclusion.** By integrating existing knowledge from epidemiological, physiological and biomechanical evidence, a theoretical causal pathway and new conceptual framework are proposed with potential implications for practice and research.

**Keywords:** conceptual framework, nursing, pressure ulcer, risk factors, tissue viability

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**Why is this research or review needed?**

- To update the pressure ulcer conceptual framework in light of recent systematic review evidence that highlighted the complex interplay of pressure ulcer risk factors.
- To link epidemiological, physiological and biomechanical evidence to facilitate increased understanding of the pressure ulcer development.

**What are the key findings?**

- The proposal of a theoretical causal pathway for pressure ulcer development that suggests direct causal factors, key indirect causal factors and other potential indirect causal factors.
- The proposal of a new pressure ulcer conceptual framework suggesting the relationships between five key biomechanical components and nine risk factors identified from epidemiological evidence and a consensus study.

**How should the findings be used to influence policy/practice/research/education?**

- The proposed conceptual framework and theoretical causal pathway could be used to underpin future research to explore the role of individual risk factors and further increase our knowledge of pressure ulcer development.
- The proposed conceptual framework and theoretical causal pathway provide the foundation for development of evidence-based pressure ulcer risk assessment in clinical practice.

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**Introduction**

Pressure ulcers are associated with ill health and poor mobility and are defined as ‘localized injury to the skin and/or underlying tissue usually over a bony prominence, as a result of pressure, or pressure in combination with shear’ (NPUAP/EPUAP 2009). They are a considerable healthcare problem worldwide (Schoonhoven et al. 2007, Vowden & Vowden 2009, Pieper 2012) in relation to the detrimental effect they have on the patients’ quality of life.
et al. 2009, 2012), as well as the financial burden to healthcare organizations (Severens et al. 2002, Bennett et al. 2004, Schuurman et al. 2009, Berlowitz et al. 2011, Dealey et al. 2012). The impact pressure ulcers have from both a quality of life and a financial perspective is influenced by their severity.

Pressure ulcers are categorized according to the international NPUAP/EPUAP (2009) classification system. Category I pressure ulcers are areas of skin redness, which do not blanch under light pressure, whereas category II pressure ulcers involve skin damage, and category III or IV pressure ulcers involve loss of fat, muscle and bone. Additional categories of unstageable (full thickness tissue loss where actual depth of the ulcer is completely obscured by slough and/or eschar) and suspected deep tissue injury (depth unknown: purple or maroon localized area of discoloured intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear) are also incorporated in the classification system (NPUAP/EPUAP 2009). To avoid the development of such lesions in clinical practice, much effort is afforded to identifying patients for whom pressure ulcer prevention interventions are needed. This is achieved by considering patient characteristics or risk factors, which predispose them to pressure ulcer development, a process known as risk assessment. As risk assessment is considered the cornerstone to prevention (AHCPR 1992, NICE 2003, NPUAP/EPUAP 2009), it is important that it is underpinned by an up-to-date conceptual framework. This paper describes the work of an international expert group and the proposal of a new pressure ulcer conceptual framework.

Background

Pressure Ulcer conceptual frameworks provide a theoretical model of the critical determinants of pressure ulcer development. This is important for both research and clinical practice. From a research perspective, pressure ulcer studies should be underpinned by a conceptual framework that is informed by evidence from all relevant fields of inquiry. This guides study aims and objectives and allows theory to be tested, to further develop the evidence base and conceptual framework. From a clinical perspective, conceptual frameworks are used to underpin pressure ulcer risk assessment guidance and tools/scales used in practice. It is, therefore, critically important that they are updated as new evidence emerges to facilitate translation of evidence into clinical care. Several pressure ulcer conceptual frameworks have been proposed over the course of the last three decades.

Braden and Bergstrom, in their conceptual model implicated intensity and duration of pressure and tissue tolerance (Braden & Bergstrom 1987). The latter related to the ability of the skin and its underlying structures to tolerate pressure without damage. It was proposed that tissue tolerance would be influenced by extrinsic and intrinsic factors. Defloor developed his conceptual scheme highlighting the importance of pressure (in the form of compressive and shearing forces), while recognizing that tissue tolerance is an important consideration (Defloor 1999). However, he viewed the latter as an ‘intermediate variable and not a causal factor’. Benoit and Mion developed their conceptual model for critically ill patients and also incorporate pressure and tissue tolerance with the latter highlighting extrinsic factors (Braden moisture and friction and shear) and intrinsic factors (metabolic supply and demand, pressure distribution capacity and threats to skin integrity) (Benoit & Mion 2012).

Another conceptual framework was proposed by NPUAP/EPUAP (2009) and underpins international guidance on the prevention and treatment of pressure ulcers. It is based on factors that influence mechanical boundary conditions and the susceptibility of the individual. The framework provides a theoretical model of the important biomechanical and physiological conditions (of both the local area and systemically), which influence the development of pressure ulcers. A summary of physiological and biomechanical evidence is described below.

Physiological and Biomechanical Evidence

The primary cause of pressure ulcers is mechanical load in the form of pressure or pressure and shear, applied to soft tissues, generally over a bony prominence (NPUAP/EPUAP 2009). Key biomechanical terms are defined in Table 1. Load that is distributed in a non-uniform or localized manner, as opposed to a uniform distribution, is potentially far more damaging to the tissues and shear forces are thought to increase tissue damage caused by pressure (Dinsdale 1974, Defloor 1999, Linder-Ganz & Gefen 2007). While it is universally recognized that both intensity and duration of pressure are of prime relevance in the development of pressure ulcers, it is difficult to determine the relative contribution of these two parameters.

Laboratory and animal studies propose several aetiological mechanisms by which stress and internal strain interact with damage thresholds to result in pressure ulcer development including localized ischaemia, reperfusion injury, impaired lymphatic drainage and sustained cell deformation (Bouten et al. 2003):

**External Mechanical Load:** comprises of all modes of external loading applied to a person’s skin as a result of contact between the skin and a support surface (including air-filled or water-filled devices that provide support) or contact between the skin of two body surfaces. The loading can be resolved into:

- Normal force: perpendicular to the skin surface; or
- Shear force: parallel to the skin surface

Pressure: normal force per unit surface area

In a clinical situation, shear forces require actual contact between the skin and the support surface, associated with normal forces, so that the skin will be exposed to a combination of both normal and shear forces.

Normal forces are distributed over the contact area, which necessitates use of the term pressure, namely normal force divided by the contact area. Shear forces are also distributed over the contact area and create external shear stresses.

**Friction:** technically, this describes all phenomena that relate to interface properties and sliding of surfaces with respect to each other (e.g. a person’s skin over clothing or bed sheets). In PU literature, the term ‘friction’ has often been defined as the contact force parallel to the skin surface in case of ‘sliding’ (i.e. sliding of surfaces along each other).

**Mechanical Boundary Condition:** the mechanical load that is applied to the skin at the interface with the supporting surface represents a boundary condition.

**Non-uniform Force:** localized to a specific area of the skin surface for which the magnitude of force may be variable.

**Deformation:** change in dimension (shape) as a result of applied loading.

**Strain:** a measure of the relative deformation.

**Stress:** force transferred per unit area. Pressure represents a special type of stress where the forces are all normal to the area over which they act.

**Morphology:** size and shape of the different tissue layers.

**Mechanical Properties of the Tissue:** refers to the stiffness and strength of the tissue material.

**Transport Properties:** refers to the rate of transport of biomolecules into/out of tissues, which may be either passive or active in nature. Active transport, which is sometimes called convection, involves metabolite transport by flow in blood and/or lymph vessels.

- Localized ischaemia: conventionally, ischaemia was thought to be the dominant aetiological factor associated with pressure ulcer development. Obstruction or occlusion of the blood vessels in soft tissues caused by external loading results in ischaemia, reduced supply of nutrients to cells and elimination of metabolites (and associated change in pH) from localized areas eventually leading to tissue damage (Kosiak 1961, Dinsdale 1974, Bader et al. 1986, Gawlitta et al. 2007).
- Reperfusion injury: during the unloading reperfusion phase, damage caused by ischaemia may be exacerbated as a direct result of the release of harmful oxygen free radicals (Peirce et al. 2000, Unal et al. 2001, Tsuji et al. 2005)
- Impaired lymphatic drainage: Occlusion of lymph vessels in soft tissues caused by external loading is associated with an accumulation of waste products and an increase in interstitial fluid contributing to pressure ulcer development (Miller & Seale 1981, Reddy et al. 1981).
- Deformation: recent studies involving, animal, engineered muscle tissue and finite element modelling have focused on the role of deformation in pressure ulcer development. These studies revealed that strains of sufficient magnitude have the potential to cause cell death over very short periods of time (Gefen et al. 2008). Gawlitta et al. (2007) considered the differences in influence of deformation and ischaemia, using tissue engineered muscle and found that deformation per se had an immediate effect, whereas hypoxia reduced cell viability over prolonged loading periods. Furthermore, animal experiments involving 2 hours of muscle compression showed that while a complete area of muscle was ischaemic, damage occurred in specific regions where high shear strain values were observed (Stekelenburg et al. 2007). Subsequent work using finite element simulations revealed that the areas of tissue damage coincided with those where the predicted strains exceeded a critical threshold (Ceelen et al. 2008). Once the critical threshold has been exceeded, the length of the exposure determined the extent of tissue damage, (Loerakker et al. 2010). Loerakker further examined the additional effects of reperfusion (Loerakker 2011). The results indicated that over short periods of loading exposure, the level of deformation was the most important factor in the damage process for muscle tissue, while ischaemia and reperfusion gradually become dominant over prolonged exposure periods. These bioengineering studies have provided important new insights into the damage thresholds for muscle tissue, but skin and fat are also implicated in pressure ulcer development.

An early pathological study identified two pathways for pressure ulcer development. This included ulcers presenting as superficial loss of the epidermis that progresses to deeper tissues if the pressure remains unrelied and deep tissue injury with necrosis of muscle and fat before destruction of the superficial layers and the appearance of a deep ulcer (Barton & Barton 1981). Bouten et al. (2003) suggest that the type of ulcer (superficial verses deep ulcer) depends on the nature of the surface loading. Superficial pressure ulcers...
are mainly caused by shear stresses in the skin layers, whereas deep ulcers are mainly caused by sustained compression of the tissues.

At the present time, there is insufficient evidence to provide definitive numerical values for the duration of pressure or damage thresholds for pressure ulcer development in a human population. The original Reswick and Rogers (Reswick & Rogers 1976) curve has been revised, as illustrated in the NPUAP/EPVAP clinical practice guideline (2009), to more accurately reflect the risk of tissue damage at the extremes of the loading periods (i.e. at very short and very long loading times). This indicates that the magnitude of pressure to induce tissue damage in the short-term is less than originally predicted by Reswick and Rogers (Linder-Ganz et al. 2006, Stekelenburg et al. 2007).

Furthermore, there is inherent variability in both individual susceptibility and local tolerance to loading parameters associated with factors including morphology and the mechanical properties of the intervening tissues. These, in turn, are affected by the patients’ characteristics, health status and exposure to specific risk factors. However, consideration of the epidemiological literature and linking of patient risk factors to the conceptual framework (NPUAP/EPVAP 2009) is not clearly articulated in the existing framework. This important omission will be addressed in this paper, to facilitate the translation of physiological and biomechanical elements to characteristics that nurses can observe in their patients. This has the potential to increase understanding and could influence risk assessment guidance and practice.

Data sources

Three sources of data were used to inform this paper. The first two sources included a systematic review of pressure ulcer risk factors (Coleman et al. 2013) and a consensus study (Coleman et al. in press). These provided the foundation for the development and validation of a new evidence-based Risk Assessment Framework (underpinned by a risk factor Minimum Data Set) for clinical practice. They were undertaken as part of a programme of work funded by the National Institute for Health Research (NIHR) regarding pressure ulcer prevention (PURPOSE: RP-PG-0407-10056). In addition, data from an expert group meeting to consider the pressure ulcer conceptual framework were also used to inform this paper.

Systematic review

The systematic review aimed to identify the factors most predictive of pressure ulcers. The approach was based on systematic review methods recommended for questions of effectiveness and adapted for risk factor studies (Cochrane 2009, CRD 2009). A full account of the method and results are reported by Coleman et al. (2013). Briefly, the search incorporated 14 electronic databases (from inception to March 2010), grey literature, contact with experts and a citation search. The search strategy was designed with guidance from the collaborative team and includes pressure ulcer search terms (Cullum et al. 2001), OVID maximum sensitivity filters for Prognosis and Aetiology or Harm and OVID maximum sensitivity filter for RCTs (CRD 2009).

No language restriction was applied. Each included study underwent quality assessment and all factors entered into multivariable modelling and those which emerged as significant were identified. Risk factors were categorized into 15 risk factor domains and 46 sub-domains and a narrative synthesis was undertaken. Evidence tables were generated for each sub-domain showing the studies where the related variable emerged as significant and those that did not.

The review included 54 eligible studies that had undertaken multivariable analyses and the narrative synthesis identified three primary risk factor domains of immobility, skin/pressure ulcer status and perfusion (including diabetes), which emerged most consistently in multivariable modelling. Important but less consistently emerging risk factor domains included nutrition, moisture, age, haematological measures, general health status, sensory perception and mental status. Only a small number of studies included body temperature and immunity and these factors require further research. Finally, there is equivocal evidence that race or gender is important to pressure ulcer development (Coleman et al. 2013).

Identifying the risk factors independently associated with pressure ulcer development, the systematic review (Coleman et al. 2013) provides a clearer understanding of the critical pressure ulcer risk factors (recognizing that some ‘important factors’ may still be lacking in confirmatory evidence due to the lack of research rather than the effect of the variable). It should also be noted that being ‘independent’ is a statistical concept and does not imply causality (Brotman et al. 2005). Although the review evidence provides good insight into the risk factors associated with pressure ulcer development at a population level, it does not fully explain the underlying pathology of pressure ulcer development. Limitations are also acknowledged. The primary studies of the review mainly observed superficial rather than severe pressure ulcers. In general, pressure ulcer risk factors were inconsistently represented in the modelling of the primary studies and a large number of potential risk factors (over 250 named variables) were used, with lack of comparable data fields for
measurement of the same construct. This limited interpretation and prevented meta-analysis to identify an item pool for a risk stratification tool. A key recommendation of the review was the development of a Minimum Data Set (MDS) for pressure ulcer research and institutional cohorts to facilitate future large-scale multivariable analyses and meta-analysis. This would underpin the development of an evidence-based pressure ulcer Risk Assessment Framework (RAF).

Consensus study

In light of the systematic review findings and limitations, a consensus study was undertaken to agree the risk factor MDS that would underpin the development of the RAF. It would identify the risk factors and assessment items important for summarizing patient risk. The consensus study was undertaken between December 2010–December 2011. A full account of the methods and results is reported elsewhere (Coleman et al. in press). In summary, the study used a modified nominal group technique based on the Research and Development/University of California in Los Angeles (RAND/UCLA) appropriateness method. It incorporated an international expert group comprising 17 clinical and academic leaders including nurses (academic and clinical nurse specialists), doctors (diabetologist, vascular surgeon, elderly care medicine and public health), bioengineers, epidemiologist and individuals with organizational development and clinical decision-making expertise.

To agree the risk factors to be included in the MDS and RAF, the expert group considered the evidence of the systematic review (Coleman et al. 2013), wider scientific evidence drawn from the expertise of the group and clinical resonance (i.e. its considered importance to clinical practice). In addition, the views of a patient and public involvement (PPI) service user group (Pressure Ulcer Research Service User Network: PURSUN) were also considered in relation to the acceptability of proposed assessment elements. During the consensus process, four levels of risk factors were identified as follows:

- Factors with strong epidemiological/wider scientific evidence and clinical resonance that increase the probability of pressure ulcer development (immobility, skin/PU status and perfusion).
- Factors with good epidemiological/wider scientific evidence and/or good clinical resonance, but showing some inconsistency in their statistical association with pressure ulcer development (albumin, sensory perception, diabetes, nutrition and moisture).
- Factors with weak or limited epidemiological/wider scientific evidence and/or clinical resonance, which could be important at an individual patient level (age, medication, pitting oedema, chronic wound, infection, acute illness and raised body temperature).
- Factors with contradictory epidemiological evidence (race and gender) or those considered to be a surrogate measure of other key risk factors (mental health, haemoglobin).

By bringing together the relevant and up-to-date fields of enquiry and clarifying key risk factors for pressure ulcer development, the consensus study highlighted the need to undertake an additional piece of work to review and enhance the pressure ulcer conceptual framework (NPUAP/EPUAP 2009).

Expert group meeting

Aim

To consider the critical determinants of pressure ulcer development to propose a new conceptual framework. The objectives were to:

- Review and update the biomechanical elements of the conceptual framework (NPUAP/EPUAP 2009).
- Propose a theoretical causal pathway for pressure ulcer development.
- Map risk factors identified in the consensus study to the updated conceptual framework.

Data collection

The expert group of the consensus study reconvened in December 2011 to address the aim and objectives detailed above. The meeting was led by two experienced facilitators and the discussions were audio-recorded and transcribed, allowing key themes to be identified. The meeting was planned, so that members had access to the outcomes of the consensus study (Coleman et al. in press), evidence of the systematic review (Coleman et al. 2013) and causal factor terminology prior to the face-to-face meeting. Familiarity with the causal factor terminology allowed us to explore the role of the risk factors in the pressure ulcer causal pathway. This was facilitated by consideration of definitions suggested by Brotman et al. (2005):

- Risk factor – a variable with a significant statistical association with a clinical outcome.
Independent risk factor – a risk factor that retains its statistical association with the outcome when other established risk factors for the outcome are included in a statistical model.

Non-independent risk factor – a risk factor that loses its statistical association with the outcome when other established risk factors for the outcome are included in a statistical model.

Brotman et al. (2005) suggests that a causal factor is a risk factor that has a causal relationship with a clinical outcome and is defined experimentally (known to affect outcome) rather than statistically. He makes a distinction between direct and indirect causal factors:

- Direct causal factor – directly impacts the outcome (or the likelihood of the outcome).
- Indirect causal factor – impacts the outcome (or affects its likelihood of occurrence) by changing a direct causal factor. If the direct causal factor is prevented from changing, then changes in the outcome will not be produced.

In our work, we further categorized indirect causal factors into key indirect causal factors (where the epidemiological/wider scientific evidence and/or clinical resonance was stronger) and other indirect causal factors.

Ethical considerations

No formal ethical scrutiny was required or undertaken for the conceptual framework expert group meeting.

Data analysis

The findings of the consensus study (Coleman et al. in press), which identified the pressure ulcer risk factors considered important for summarizing patient risk, provided the initial framework to address the study aims. In addition, the researcher (SC) listened to the audio-tapes of the conceptual framework expert group meeting discussions and read the associated transcripts in total to ensure completeness. The analysis provided the basis for the new proposed pressure ulcer conceptual framework and theoretical causal pathway.

Validity and reliability

Validity and reliability issues relating to this study are linked to the preceding consensus study (Coleman et al. in press). While it is acknowledged that it is difficult to assess the validity of consensus judgements at the time the judgements are made (Black et al. 1999), the consensus study applied principles of good practice in the planning and delivery of the consensus process as previously reported (Coleman et al. in press). This included the involvement of a mixed-speciality expert group (Hutchings & Raine 2006) and consideration of relevant evidence throughout the process. Following analysis of the conceptual framework meeting, the researcher (SC) drafted the new proposed pressure ulcer conceptual framework and theoretical causal pathway and circulated this to the expert group via email to ensure content validity. This led to minor revisions of the work.

Results

Revised NPUAP/EPWAP conceptual framework (2009)

The in-depth discussions of the expert group led to amendments to the existing NPUAP/EPWAP conceptual framework (2009), as detailed in Figure 1. Most notably, it was recognized that while mechanical properties of the tissues and geometry (morphology) of the tissues and underlying bones have an impact on the internal strains and stresses (as an example, subjects who are either very emaciated or very obese will have enhanced strains and stresses in the soft tissues), its impact was considered to be more relevant to the susceptibility of the individual, i.e. having an impact on the damage threshold and so was moved as detailed in Figure 1. Furthermore, transport (perfusion and lymphatic drainage) also has an impact on the damage threshold of the individual and this would be affected by temperature in terms of vasodilation/vasoconstriction, thereby affecting tissue perfusion. The underlying physiology of an individual will also have an impact on their repair capacity and this was an important consideration that was captured in the amended conceptual framework (Figure 1). The amended conceptual framework and its key components provided the foundation on which to link to the epidemiological evidence.

Theoretical causal pathway

The proposed causal pathway for pressure ulcer development detailing the direct, key indirect and other potential indirect causal factors is illustrated in a theoretical schema (Figure 2). Table 2 shows the mapping of the direct causal factors and key indirect causal factors against the key components of the enhanced NPUAP/EPWAP (2009) conceptual framework. Although it was recognized that the presence and weighting of specific risk factors may vary in
relation to the anatomical site of the pressure ulcer, it was not possible to delineate the evidence to skin site level risk factors.

**Direct causal factors**

Three characteristics were classified as direct causal factors including immobility, skin/pressure ulcer status (incorporating existing and previous pressure ulcer and general skin status) and perfusion. Immobility is a necessary condition for pressure ulcer development and, through its affect on mechanical boundary conditions (Table 2), has a direct impact on the outcome (or the likelihood of the outcome). It is, therefore, considered a direct causal factor (Figure 2). Of note is that friction and shear is not specified as a patient characteristic, rather it is a

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**Figure 1** Enhancement of NPUAP/EPUAP (2009) factors that influence susceptibility for pressure ulcer development.

**Figure 2** Theoretical schema of proposed causal pathway for pressure ulcer development. The solid arrows show the causal relationship between the key indirect causal factors and direct causal factors and the outcome. Interrupted arrows show the causal relationship between other potential indirect causal factors and key indirect causal factors and between direct causal factors. Interrupted arrows also demonstrate interrelationships between direct causal factors and indirect causal factors.
Identifying whether skin/pressure ulcer status and poor perfusion represent direct or indirect risk factors is less straightforward. It could be assumed that they are indirect factors as without some degree of immobility, a pressure ulcer would not develop. However, this is not in keeping with the definitions of causal factors detailed above. Furthermore, it oversimplifies the complex interplay of factors required to lead to tissue damage. There is strong epidemiological/wider scientific evidence that poor perfusion and skin/pressure ulcer status reduce patients’ tolerance to pressure and increase the likelihood of pressure ulcer development. This suggests that they are direct causal factors and may explain why some immobile patients develop pressure ulcers while others do not.

Further insight was gained by mapping skin/pressure ulcer status and poor perfusion to the conceptual framework and it was apparent that they were clearly implicated in the susceptibility and tolerance aspect of the framework (Table 2).

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Mechanical Boundary Conditions: Type of loading (shear, pressure, friction) &amp; magnitude &amp; duration of mechanical load</th>
<th>Individual Geometry (Morphology) of the tissue &amp; bones</th>
<th>Individual Mechanical Property of the Tissues</th>
<th>Individual Transport &amp; Thermal Properties</th>
<th>Individual Physiology &amp; Repair</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immobility</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skin/PU Status</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Poor Perfusion</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poor Nutrition</td>
<td>(x) in extreme cases</td>
<td>(x) in extreme cases</td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Moisture</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poor Sensory Perception &amp; Response</td>
<td>(x) through immobility</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>(x) through sensory perception</td>
<td></td>
<td></td>
<td></td>
<td>(x) through perfusion</td>
</tr>
<tr>
<td>Low Albumin</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(x) through perfusion</td>
</tr>
</tbody>
</table>

Characteristic of the mechanical boundary condition (Table 2).

Key indirect causal factors

Moisture, sensory perception, diabetes, low albumin and poor nutrition were considered key indirect causal factors, as they have an impact on the outcome (or affect its likelihood of occurrence) by changing a direct causal factor (Figure 2).

Other potential causal factors

The theoretical conceptual schema (Figure 2) was further developed to include other indirect causal factors to illustrate the potential relationships and impact of diverse factors that may be involved in the causal pathway. However, it is recognized that the interrelationships among potential and key indirect causal factors are complex and require further elucidation. Other indirect causal factors include those with weak or limited epidemiological/wider scientific evidence, but are thought to have an impact on key indirect and direct causal factors. They include age, medication, pitting oedema and other factors relating to general health status including infection, acute illness, raised body temperature and chronic wound.
New proposed pressure ulcer conceptual framework

Following consideration of the causal pathway for pressure ulcer development (Figure 2) and mapping of direct and key indirect causal factors for pressure ulcer development against the components of the enhanced conceptual framework (Table 2), a new conceptual framework (Figure 3) is proposed. This enables the epidemiological evidence to be linked to the physiological and biomechanical elements of the conceptual framework. The new framework proposes the relationship between the mechanical boundary conditions and the susceptibility and tolerance of the individual. The risk factors that have an impact on the mechanical boundary conditions and the susceptibility and tolerance of the individual are detailed in the framework and are based on the direct causal factors including immobility, skin/pressure ulcer status and poor perfusion, as well as the key indirect causal factors of poor sensory perception and response, diabetes, poor nutrition, moisture and low albumin. For simplicity, the risk factors are represented under the elements they are thought to predominantly affect (either mechanical boundary conditions or susceptibility and tolerance of the individual). However, the interrupted line running under the risk factors indicates that some risk factors may have an effect on both sides of the framework, which is more clearly articulated in the theoretical schema (Figure 2) and risk factor mapping (Table 2). The absence of risk factors on either the individual susceptibility and tolerance or the mechanical boundary conditions side of the framework would affect the likelihood of pressure ulcer development, i.e. a patient with good perfusion may be able to tolerate higher levels of immobility (without developing a pressure ulcer) than someone with poor perfusion.

Discussion

This new proposed pressure ulcer conceptual framework incorporates key physiological and biomechanical components and their impact on internal strains, stresses and damage thresholds. Direct and key indirect causal factors suggested in a theoretical causal pathway are mapped to the physiological and biomechanical components of the framework. Agreeing the proposed elements of the new conceptual framework proved challenging as while the physiological and bioengineering research, the systematic review and the outcomes of the consensus study (Coleman et al. in press) provide a good starting point, there are still many gaps in the evidence base. In addition, the proposal of a causal pathway for any condition/disease is a complicated process. For simplicity, the pathway detailed in this paper only considers a one-directional relationship between risk factors but, in reality, bi-directional relationships exist and causal factors may have multiple roles in a pathway (e.g. moisture has an impact on the vulnerability of the skin and may also effect the impact of immobility by increasing the likelihood of friction and shear).

It should be noted that the new conceptual framework does not consider varying parameters of risk factors (e.g. patients have varying levels of mobility, nutrition, moisture, etc.) in the causal pathway and how these have an impact on pressure ulcer outcome. Furthermore, it does not explain how varying combinations of risk factors increase the likelihood of pressure ulcer development. The importance of individual risk factors may also vary in relation to body site, for example a patient with peripheral vascular disease may have reduced tolerance to pressure to their heels, but not to their trunk areas. Patients may also have conditions such as contractures, which may increase their risk of pressure ulcers at less commonly encountered body sites. In addition, the new conceptual framework does not clearly articulate the aetiological mechanisms of importance for risk factors, for example there is still uncertainty about the specific mechanisms of importance relating to perfusion.

Limitations of the approach relate to the uncertainties associated with the primary research considered in the consensus study (Coleman et al. in press) and in the proposal of the new conceptual framework. The bioengineering research is limited due to its development in animal or tissue-engineered muscle models as opposed to human subjects. The evidence of the systematic review is limited by poor reporting, heterogeneity of patient populations, inconsistent inclusion of pressure ulcer domains, inconsistent measurement of risk factor variables, the use of different outcomes and lack of differentiation between pressure ulcer sites. Furthermore, the primary studies of the systematic review mainly observed superficial pressure ulcers, while much of physiological and bioengineering research relates to muscle tissue and it could be argued that the associated aetiological mechanisms differ. However, there is no evidence that the key direct causal factors for superficial or deep pressure ulcers are different, rather it is the nature of surface loading that influences the type of pressure ulcer that develops (i.e. initially developing superficially or in muscle tissue) (Bouten et al. 2003).

Implications for nursing

The new conceptual framework and theoretical causal pathway together propose clearer linkage between the physiological and biomechanical determinants of pressure ulcer
development and patient risk factors. They provide a framework for understanding the critical determinants of pressure ulcer development and facilitate the translation of physiological and biomechanical elements to characteristics that nurses can observe in their patients. They could lead to increased understanding and have the potential to influence risk assessment guidance and practice.

The proposed conceptual framework and theoretical causal pathway also have implications for research. They provide an up-to-date account of how existing evidence can be used to develop theory and help to identify gaps in our knowledge base. These could be used to underpin and guide future research, building on the evidence and enabling us to more clearly define the role of individual pressure ulcer risk factors conceptually and operationally.

**Conclusion**

This paper describes work undertaken by an international expert group and the proposal of a new pressure ulcer conceptual framework. The approach incorporated consideration of physiological, biomechanical and epidemiological evidence, as well as the outcomes of a consensus study and the views of an expert panel. This was enabled by consideration and enhancement of the NPUAP/EPUAP (2009) conceptual framework, the proposal of a theoretical causal pathway for pressure ulcer development and mapping of risk factors to the conceptual framework. The new conceptual framework and theoretical causal pathway propose the critical determinants of pressure ulcer development and could influence risk assessment guidance and practice. They could also be used to underpin and guide future pressure ulcer research, to further explore the relationship between risk factors and increase our understanding of pressure ulcer development.

**Funding**

This publication presents independent research funded by the National Institute for Health Research (NIHR) under its Programme Grants for Applied Research Programme (RP-PG-0407-10056). The views expressed in this publication are those of the author(s) and not necessarily those of the NHS, the NIHR or the Department of Health.

**Conflict of interest**

No conflict of interest has been declared by the author(s).
Author Contributions

The authors have confirmed that all authors meet the IC-MJE criteria for authorship credit (www.icmje.org/ethical_1author.html), as follows:

- substantial contributions to conception and design of, or acquisition of data or analysis and interpretation of data,
- drafting the article or revising it critically for important intellectual content, and
- final approval of the version to be published.

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