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Care Delivery

Exploring the gap between sensor-detected (SDH) and person-reported hypoglycaemia (PRH): The voice of people living with diabetes

Monika Cigler[1] | Omaima El-Hakouni[1] | Renald Mecani[1] | François Pouwer[2,3,4] | Evertine J. Abbink[5] | Michaela Moser[6] | Bastiaan E. de Galan[5,7,8] | Eric Renard[9] | Mark Evans[10,11] | Julie Maria Bøggild Brøsen[12] | Ulrik Pedersen-Bjergaard[12] | Rory J. McCrimmon[13] | Simon Heller[14] | Petra M. Baumann[1] | Uffe Søholm[2,15,16] | Silvia Basta[1] | Patrick Divilly[17] | Natalie Zaremba[18,19,20] | Stephanie A. Amiel[18] | Pratik Choudhary[21] | Julia K. Mader[1] | on behalf of the Hypo-RESOLVE consortium[†]

Correspondence
Julia K. Mader, Division of Endocrinology and Diabetology, Department of Internal Medicine, Medical University of Graz, 8036 Graz, Austria.
Email: julia.mader@medunigraz.at

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Abstract

Background and Aims: In people with insulin-treated diabetes experiencing hypoglycaemia, the multicentre Hypo-METRICS study found that 60% of sensor-detected hypoglycaemic episodes (SDH) were asymptomatic, and over 40% of person-reported hypoglycaemia (PRH) occurred at glucose levels ≥70 mg/dL (3.9 mmol/L). This subanalysis explored participants' experiences of these episodes to identify possible clinical implications.

Methods: Fifty-eight Austrian participants received a 15-item questionnaire on their experience of asymptomatic hypoglycaemia and symptoms at glucose levels ≥70 mg/dL (3.9 mmol/L).

Results: The response rate was 86% ($n = 50$). Among all participants, 56% ($n = 28$) reported experiencing hypoglycaemic symptoms at glucose levels ≥70 mg/dL (3.9 mmol/L) "sometimes" or "often." They attributed this to a combination of a threshold shift due to chronic hyperglycaemia, rapid glucose decline and fear of hypoglycaemia. 68% of all SDH < 70 mg/dL and 59% of those below the clinically critical level of 54 mg/dL were asymptomatic.

Conclusion: These results demonstrate that SDH and PRH each capture different, yet equally important, dimensions of the hypoglycaemia experience. Relying on only one source of information inevitably provides an incomplete picture. By integrating the patient's voice, diabetes professionals can provide appropriate

[†]See "Funding" section for details.

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support and tailor continuous glucose monitoring (CGM) alarm settings and treatment plans to truly meet individual needs.

KEYWORDS

continuous blood glucose monitoring, diabetes, patient experience, type 1 diabetes, type 2 diabetes

1 | INTRODUCTION

Hypoglycaemia remains one of the main limiting factors in achieving optimal glycaemic control in people with diabetes. Despite major advances in diabetes technology and the widespread use of continuous glucose monitoring (CGM), which can reduce hypoglycaemia risk,[1–8] hypoglycaemic episodes continue to impair quality of life, cognitive performance, and treatment adherence.[9] In 2017, the International Hypoglycaemia Study Group defined three clinically relevant levels of hypoglycaemia[1]: level 1 (glucose <70 mg/dL (3.9 mmol/L) but ≥54 mg/dL (3.0 mmol/L)) marks an alert threshold, level 2 (glucose <54 mg/dL or 3.0 mmol/L) denotes clinically significant hypoglycaemia requiring prompt action, and level 3 refers to severe hypoglycaemia associated with cognitive impairment that requires assistance.[10]

With the increasing use of CGM, it has become evident that many sensor-detected hypoglycaemic (SDH) episodes are not accompanied by symptoms, while person-reported hypoglycaemia (PRH) sometimes occurs at glucose levels above the glycaemic threshold denoting level 1 episodes.

The multicentre HypoMETRICS study,[11] conducted as part of the EU Horizon 2020 Hypo-RESOLVE project,[12] demonstrated that over 60% of CGM-detected hypoglycaemic episodes were asymptomatic– irrespective of clinical state of hypoglycaemia awareness– and that more than 40% of PRH occurred at glucose levels exceeding 70 mg/dL (3.9 mmol/L).[13] These findings highlight a striking discrepancy between SDH and PRH, raising important questions about the perception, reporting, and clinical interpretation of hypoglycaemia in daily life.

Explaining the reasons for this low concordance between SDH and PRH requires greater understanding of how people with T1D and T2D react practically and emotionally to asymptomatic episodes when alerted by glucose monitoring technology as well as symptoms that occur when the glucose levels are not hypoglycaemic. This will help diabetes professionals provide appropriate support during consultations and when planning future research.

The present study explored participants' experiences, interpretations, and perceptions of hypoglycaemia following their participation in the Hypo-METRICS study, aiming to identify factors from the patients' perspective that may have contributed to the observed discrepancies between SDH and PRH.[14]

What's new?

- A substantial discrepancy exists between sensor-detected hypoglycaemia (SDH) and person-reported hypoglycaemia (PRH), with many SDH episodes being asymptomatic and many PRH episodes occurring at glucose levels ≥70 mg/dL.
- Over half of participants reported hypoglycaemic symptoms at non-hypoglycaemic glucose levels, likely due to factors such as rapid glucose decline, chronic hyperglycaemia, and fear of hypoglycaemia.
- A large proportion of SDH, including clinically significant episodes (<54 mg/dL), occurred without symptoms, indicating under-recognition of hypoglycaemia.
- SDH and PRH capture different but complementary aspects of the hypoglycaemia experience and should both be considered in clinical practice.
- Incorporating patient perspectives can improve individualized care, including tailored CGM alarm settings and treatment strategies.

2 | METHODS

2.1 | Participants

This questionnaire study was completed at the Austrian site of the HypoMETRICS study, which recruited adults with insulin-treated diabetes and at least one hypoglycaemic episode in the previous 3 months (either measured by sensor or glucose meter or symptomatic) into a 10 week observation period. During this period, they wore a blinded CGM (a Freestyle Libre 2 in addition to their usual monitoring) and an activity monitor and used a purpose-built

mobile phone app to record details of any hypoglycaemia episode that occurred during the 10 weeks.[11]

All Austrian participants who completed the HypoMETRICS study (98 out of 602 across all centres) and consented to be recontacted ($n = 58$) were invited to complete a follow-up questionnaire (6–10 months after completing the HypoMETRICS study) to explore reasons behind the previously observed low concordance between SDH (as measured by the blinded study CGM) and PRH (as recorded in the purpose-built mobile phone app). The last questionnaire was completed and sent back 11 months after that participant had finished HYPOmetrics. The study included individuals with both type 1 (T1D) and type 2 diabetes (T2D) from various age groups; approximately one-quarter of the participants had impaired awareness of hypoglycaemia (IAH) according to the GOLD score.[15] On average, participants' baseline glucose levels were higher than those recommended in the International Consensus on Use of Continuous Glucose Monitoring.[16]

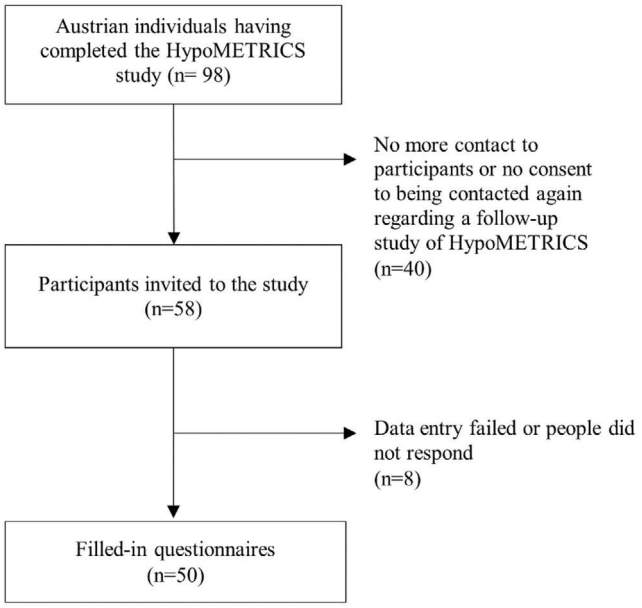

FIGURE 1 Study participation flow.

2.2 | Ethical approval and informed consent

Ethical approval for the study was granted by the Ethics Committee of the Medical University of Graz and all procedures were carried out in compliance with the Declaration of Helsinki and the principles of Good Clinical Practice. Written informed consent was obtained from all participants before they received access to the survey link. For participants unfamiliar with electronic communication or email, the questionnaire could be completed on-site or by telephone.

An overview of the participant inclusion process is presented in Figure 1.

2.3 | Study design and questionnaire description

The questionnaire was thematically structured into two main sections:

a. Symptoms without confirmed hypoglycaemia: In the first section, participants were asked to reflect on episodes in which they had reported hypoglycaemic symptoms despite glucose values being above 70 mg/dL. The items focused on potential explanations for experiencing such symptoms at non-hypoglycaemic levels, the specific contexts or situations in which these episodes occurred, participants' personal interpretations of these experiences, and their perceived fear of hypoglycaemia.
b. Asymptomatic SDH: These questions asked participants to describe typical situations in which such episodes occurred and to share their personal hypotheses regarding possible underlying causes. Asymptomatic hypoglycaemia was defined as SDH that did not show any PRH within 1 h after the start of the SDH.

Most participants had worn their own unblinded CGM in addition to the blinded study CGM during the HypoMETRICS study. They were therefore aware both of the glucose levels at which their symptoms occurred and of hypoglycaemic episodes that presented without any symptoms. Those without an unblinded CGM answered based on their personal experience and awareness of their diabetes. (These persons all reported occasionally verifying their glucose values with SMBG, which also helped them develop a sense of how often asymptomatic episodes occurred).

Concerning the blinded sensor used, manufacturer data indicate that, in the lower measurement range, approximately 98.4% of Libre 2 readings are within ±1.1 mmol/L (≈ ± 20 mg/dL) of reference values. A hypoglycaemic episode recorded by CGM was defined as spending at least 15 min below threshold, with resolution of hypoglycaemia above the threshold for 15 min.

2.4 | Additional participant feedback and clarification procedures

Although the questionnaire consisted primarily of structured, closed-ended items, participants were explicitly encouraged to provide additional comments or questions

throughout the study via email, telephone, or in person. As the primary point of contact for all 50 participants also during HypoMETRICS at the Austrian site, the first author received substantial supplementary feedback during and after the study. This included explanations of individual responses, clarifying questions about the meaning of specific items, and spontaneous reflections regarding hypoglycemia experiences. This additional feedback served an important function in ensuring that questionnaire responses accurately reflected participants' intended interpretations. In cases where an item appeared potentially ambiguous—particularly when participants selected "yes" on questions that could be interpreted in multiple ways—the first author contacted respondents individually to verify their understanding of the item and the reasoning behind their chosen response. These clarifications were used to avoid misclassification due to misunderstood items and to ensure that participants' responses genuinely represented their own perceptions and interpretations of their hypoglycaemia experiences. Given this context, the conclusions presented in the manuscript are based on the questionnaire responses themselves, supported by the assurance—derived from follow-up clarifications where relevant—that participants' selections aligned with their intended meaning.

2.5 | Data handling and statistics

All completed questionnaires were processed and securely managed by the research team at the Division of Endocrinology and Diabetology, Medical University of Graz. Data collection complied with the General Data Protection Regulation (GDPR). Only authorized team members had access to anonymized data. Responses were transferred to an Excel database, checked for completeness and consistency, and prepared for analysis.

Descriptive statistical methods using Excel were employed to summarize participant characteristics and the main questionnaire outcomes. The analysis was primarily exploratory in nature, aiming to provide a detailed overview of the dataset rather than to perform hypothesis testing or apply advanced inferential techniques. Open-ended responses were analysed qualitatively using thematic analysis to identify common themes and illustrative examples.

3 | RESULTS

3.1 | Participant demographics and response rate

Out of the 58 questionnaires distributed, responses from 50 participants (86%) were considered valid and were included in the analysis. Eight responses were excluded due to missing ID numbers or because they contained insufficient data (defined as completion of less than 50% of the questionnaire items). As not all participants answered every question, the number of valid responses varies across items, and percentages are based on the number of participants who responded to each question.

TABLE 1 Participants' demographic characteristics.

Age (mean ± SD)	56.6 ± 15.4 years
Male gender, *n* (%)	28/50 (56%)
T1D (diabetes mellitus, type 1)	24 (48%)
T2D (diabetes mellitus, type 2)	26 (52%)
IAH (impaired awareness of hypoglycaemia) by Gold score, *n* (%)	13 (27%), average Gold score 4.5
TIR	67.5 ± 20.3%
TBR	3.7 ± 5.7%
TAR	28.2 ± 19.8%
GMI	7.1 ± 1.0%
Diabetes duration	18.41 ± 9.71 years
Baseline T1D	
TIR	61.67 ± 18.00%
TBR	6.33 ± 7.08%
TAR	31.58 ± 19.21%
Age (mean ± SD)	38.46 ± 14.59 years
Male gender, *n* (%)	10 (41.7%)
Diabetes duration	15.58 ± 9.41 years
Baseline T2D	
TIR	72.96 ± 21.07%
TBR	1.35 ± 2.06%
TAR	25.00 ± 20.24%
Age	64.00 ± 11.48 years
Male gender, *n* (%)	18 (69.2%)
Diabetes duration	21.50 ± 9.26 years

For baseline characteristics of the participants see Table 1 (Participants' demographic characteristics).

3.2 | Hypoglycaemic symptoms at glucose levels ≥70 mg/dL

A total of 28 respondents (56%; T1D = 14, T2D = 14) reported that they "sometimes" or "often" experienced symptoms of hypoglycaemia at glucose levels ≥70 mg/dL. When asked to describe their symptoms, two participants reported neuroglycopenic symptoms including visual disturbances, with one of them also experiencing speech difficulties and dizziness. A further 33 participants (this question was also posed to those participants

TABLE 2 Hypoglycaemia symptoms ≥70 mg/dL (3.9 mmol/L) – subgroup differences.

Parameter	Symptoms "Sometimes/Often" ($n = 28$)	Symptoms "Never/Rarely"
HbA1c	7.6 ± 1.30%	6.7 ± 0.6%
Diabetes duration	18.16 years	18.70 years
age	43 ± 17 years	55 ± 21 years
Time In Range (TIR)	61.2 ± 22.7%	74.9 ± 13.5%
Time Above Range (TAR)	33.8 ± 22.1%	19.7 ± 12.5%
Time Below Range (TBR)	2.6 ± 3.9%	5.5 ± 7.5%
SDH	episodes (10 weeks) 43 ± 40	episodes (10 weeks) 64 ± 47
PRH	episodes (10 weeks) 30 ± 35	episodes (10 weeks) 32 ± 26
Number of PRH episodes that were not reflected in the SDH data	14 ± 20	12 ± 11
Impaired Awareness of Hypoglycaemia (IAH)	12%	15%

that answered they "rarely" experienced symptoms under non-hypoglycaemic conditions) described symptoms such as trembling, cold sweats, hunger, and difficulty concentrating, which they reported as "mild".

When comparing the 28 participants who frequently reported symptoms ("sometimes/often") with those who reported them "never/rarely", the following differences in glycaemic parameters were observed (Table 2):

The group reporting frequent symptoms at high glucose levels was younger and had a higher mean HbA1c, with lower TIR and TBR.

A comparison between T1D and T2D participants among those participants that experienced frequent hypoglycaemic symptoms in the non-hypoglycaemic range showed that HbA1c was comparable in both groups, with TAR and TBR showing higher values in T1D. Diabetes duration was lower in T1D, and the number of SDH and PRH was four times higher than in T2D (for detailed analyses see Table S1).

When asked about possible explanations for experiencing symptoms at non-hypoglycaemic glucose levels, 11 participants (28%; T1D/T2D = 7/4) considered it possible that their symptoms were caused by fear of hypoglycaemia rather than by true hypoglycaemia. In contrast, 17 participants (44%; T1D/T2D = 7/10) were confident that these symptoms represented genuine hypoglycaemic episodes despite occurring at non-hypoglycaemic levels; most of them attributed their symptoms to a rapid decline in blood glucose.

Commonly reported situations in which participants described experiencing these symptoms at non-hypoglycaemic levels included: (1) periods of stress or (2) physical exertion, (3) states of fatigue or nervousness, (4) low blood pressure (in some persons objectively measured, but in others only assumed due to feelings of dizziness or low energy), (5) during fasting or dietary restriction, (6) during perceived hormonal fluctuations due to menopause/the menstrual cycle, and (7) after prolonged absence of prior hypoglycaemic episodes.

3.3 | Asymptomatic hypoglycaemia

The 50 participants reported an average of 33 ± 35 PRH episodes (at any glucose value and not significantly different for persons with IAH). Continuous glucose monitoring identified an average of 54 ± 45 SDH episodes per participant over the 10 weeks data collection. Average coefficient of variability of all participants was 35%.

In total, 68% of all SDH episodes below 70 mg/dL (3.9 mmol/L) and 59% of those below 54 mg/dL (3.0 mmol/L) were not matched by a PRH. Also, participants' responses to our questionnaire did not correspond strongly to SDH data, indicating that low glucose levels often occurred without being noticed by the participants (see Table 3).

Situations in which asymptomatic hypoglycaemia commonly occurred included periods of physical inactivity, evening hours, and during phases of intense concentration or distraction.

4 | DISCUSSION

In our survey, we explored the gap between SDH and PRH based on insights from 50 Austrian participants from

TABLE 3 Participants' self-reported perception (questionnaire) and SDH.

Participants' self-reported perception (questionnaire)	CGM data: proportion of SDH episodes that were asymptomatic (=not matched with PRH)
When my glucose drops below 70 mg/dL (3.9 mmol/L), I *always* have symptoms ($n = 12$).	65% of all hypoglycaemic episodes in this group were asymptomatic
When my glucose drops below 70 mg/dL (3.9 mmol/L), these episodes are *rarely asymptomatic* ($n = 20$).	70% of all hypoglycaemic episodes in this group were asymptomatic.
When my glucose drops below 70 mg/dL (3.9 mmol/L), these episodes are *sometimes/often asymptomatic* ($n = 15$).	67% of all hypoglycaemic episodes in this group were asymptomatic.

the Hypo-METRICS study. Besides confirming patterns similar to the main Hypo-METRICS findings, our study provided perspectives on participants' perceived reasons for discrepancies between SDH and PRH as well as their experiences during these events.

The fact that over half of our participants reported experiencing hypoglycaemic symptoms at glucose levels ≥70 mg/dL (3.9 mmol/L) "sometimes" or "often" was consistent with findings of the HypoMETRICS study. However, while Divilly et al.[11] reported no association between HbA1c and the proportion of PRH without corresponding SDH, our subgroup showed an indication of a weak trend toward such a relationship (see Table 2).

Literature confirms that individuals with chronically high glucose levels can experience a physiological threshold shift, causing symptoms at non-hypoglycaemic values.[17,18] Also – although the literature is somewhat inconsistent on this point - there are studies that show that during decrements in plasma glucose concentration, symptoms of hypoglycaemia may occur at higher glucose concentrations in patients with poorly controlled insulin-dependent diabetes.[19] This aligns with reports from many of our participants, who attributed their hypoglycaemic symptoms in the non-hypoglycaemic range to rapid glucose drops.

Age also appeared to differ between groups, with participants reporting frequent symptoms being younger on average (43 ± 17 years) than those with no or only rare symptoms (55 ± 21 years). From the comparison between T1D and T2D in this group we can see that people with T1D in our population experience greater glucose variability and more frequent true hypoglycaemia despite having similar HbA1c levels. Nevertheless, the high TAR observed in both groups suggests that chronic hyperglycaemia—and the associated upward shift in symptom thresholds—may be an important contributor to hypoglycaemic symptoms occurring above the hypoglycaemia threshold in both T1D and T2D.[20]

Moreover, psychological factors also seem to have played a role in our population, as 28% of participants indicated that their symptoms might have been related to fear of hypoglycaemia rather than to true hypoglycaemia. Interestingly, all these participants used an (unblinded) CGM device and could therefore see their glucose decreasing– whereas none of the participants without an unblinded CGM device reported fear of hypoglycaemia as a potential explanation for their symptoms.

In contrast, our data also suggest that SDH (<70 mg/dL (3.9 mmol/L)) are frequently not recognized by people with T1D or T2D. The episode rates of asymptomatic hypoglycaemia as opposed to PRH in this subanalysis were similar to those reported for the Hypo-METRICS cohort as a whole and are also in line with other studies.[21,22] Søholm et al.[23,24] demonstrated that even simply knowing of a glucose value below 70 mg/dL (3.9 mmol/L) negatively affects daily functioning– the impact is the same regardless of whether the person actually experiences hypoglycaemic symptoms, merely sees the low value on their CGM device (nocturnal SDH may simply have been due to sensor compression during sleep) or only takes preventive measures without actually having a hypoglycaemic episode. These findings suggest that alerts for glucose values just below 70 mg/dL (3.9 mmol/L) – defined by the IHSG as level 1 hypoglycaemia and intended as an early warning to prompt action rather than being an indicator of a clinically significant risk – may impose an unnecessary burden on patients when such values are asymptomatic. (Physiological harmlessness of these episodes was not specifically assessed in this study. However, these values can also be found in healthy individuals without diabetes). Personalised CGM alarm settings – tailored to individual thresholds and the duration of clinically significant hypoglycaemia may offer a more appropriate approach.[25]

Our data also showed that 59% of asymptomatic hypoglycaemic episodes (SDH) occurred at glucose levels below 54 mg/dL (3.0 mmol/L), which– even without symptoms– is considered clinically significant and requires immediate attention.[10] It is at this stage that neuroglycopenia can occur when the cerebral cortex is no

longer adequately supplied with glucose, which may lead to impaired cognitive function.[26] Repeated episodes of level 2 hypoglycaemia can also lead to IAH.[27,28] A Hypo-RESOLVE clamp study demonstrated that level 2 hypoglycemia consistently deteriorated cognitive performance to a largely similar extent in all types of diabetes as well as in healthy controls.[29]

However, major analyses estimating the risk of long-term consequences (such as death or acute cardiovascular disease) relied on data collected primarily through SMBG and symptomatic reporting, not CGM. As they may therefore have missed a substantial proportion of asymptomatic hypoglycaemia, the calculated hazard ratios for mortality and cardiovascular disease should, according to the authors of these studies, not be assumed to apply to asymptomatic hypoglycaemia. At the same time, these analyses suggest that the cumulative number of hypoglycaemia episodes—rather than TBR—is the parameter most strongly associated with these adverse outcomes, particularly in people with Type 2 diabetes.[30]

A major strength of our study is that all 50 participants had previously taken part in the Hypo-METRICS study and had, therefore, been focusing on their hypoglycaemic episodes and the role these episodes played in their daily functioning for 10 weeks already. As a result, participants were highly engaged and motivated to provide feedback. Moreover, the first author had served as the primary point of contact since the start of Hypo-METRICS, facilitating open communication and enabling the collection of both positive and critical feedback. Comprehensive medical and demographic data for all participants had already been collected as part of the original study and were readily available for reference.

Our study is not without limitations.

First, only 50 participants were included, both T1D and T2D, who were selected from the Austrian subjects of the Hypo-METRICS trial. Some of the subgroups, therefore, were too small to allow for meaningful subgroup analyses. Also, including a control group of persons without diabetes who reported "hypoglycemia-like" symptoms on a daily basis could have helped differentiate true hypoglycemia experiences from normophysiological sensations.

Second, this study was undertaken a number of months after the HypoMETRICS study and so results may have some recall bias and may include some participants' current experience of CGM and hypoglycaemia rather than experience during HypoMETRICS.

Third, reliability analyses (e.g., Cronbach's alpha) were not conducted, as the primary aim of this study was exploratory and the questionnaire was specifically designed for this context. However, the instrument was pilot-tested with ten participants, and their feedback was used to refine the items and improve clarity and relevance. This preliminary step supports the content validity of the instrument. Future research should include formal psychometric testing to further establish reliability and validity.

5 | CONCLUSION

Our findings underscore the complexity of the relationship between SDH and PRH. Participants' feedback suggests that symptoms occurring at glucose levels above the clinical threshold of 70 mg/dL (3.9 mmol/L) may often reflect a combination of physiological responses, such as the effects of chronically elevated glucose levels or rapid glucose declines, alongside psychological responses, such as the fear of hypoglycaemia, when observing falling glucose levels on CGM devices. Conversely, a large proportion of SDH below 70 mg/dL (3.9 mmol/L) remains substantially under-recognized, with 59% of episodes below the clinically significant level of 54 mg/dL occurring without being matched by a PRH.

These results demonstrate that SDH and PRH each capture different, yet equally important, dimensions of the hypoglycaemia experience. Relying on only one source of information inevitably provides an incomplete picture. Thus, both SDH and PRH should be systematically documented in clinical practice and research to obtain a more comprehensive understanding of the real-world burden of hypoglycaemia. Also, an understanding of the patient's perspective—recognizing both SDH and the subjective emotional and functional disruption of daily life (PRH)—must be high on the agenda during clinical consultations. By integrating the patient's voice, diabetes professionals can provide appropriate support and tailor CGM alarm settings and treatment plans to truly meet individual needs.

AFFILIATIONS

[1]Division of Endocrinology and Diabetology, Department of Internal Medicine, Medical University of Graz, Graz, Austria

[2]Department of Psychology, University of Southern Denmark, Odense, Denmark

[3]Steno Diabetes Center Odense (SDCO), Odense, Denmark

[4]Department of Health and Caring Sciences, Western Norway University of Applied Sciences, Bergen, Norway

[5]Department of Internal Medicine of Radboud University Medical Centre, Nijmegen, the Netherlands

[6]Department for Applied Computer Sciences/Business Informatics, FH Joanneum, Graz, Austria
[7]Department of Internal Medicine, Maastricht University Medical Center, Maastricht, the Netherlands
[8]CARIM Cardiovascular Research Institute Maastricht, Maastricht University, Maastricht, the Netherlands
[9]Montpellier University Hospital, Montpellier, France
[10]Institute of Metabolic Science and Department of Medicine, University of Cambridge, Cambridge, UK
[11]Level 4, Institute of Metabolic Science, Box 289, Addenbrooke's Hospital, Cambridge, UK
[12]Department of Endocrinology and Nephrology, Copenhagen University Hospital, Hillerød, Denmark
[13]School of Medicine, University of Dundee, Dundee, UK
[14]Division of Clinical Medicine, School of Medicine and Public Health, University of Sheffield, Sheffield, UK
[15]School of Psychology, Institute for Health Transformation, Deakin University, Geelong, Victoria, Australia
[16]The Australian Centre for Behavioural Research in Diabetes, Diabetes Victoria, Carlton, Victoria, Australia
[17]St. Vincent's University Hospital, Dublin, University College Dublin, Dublin 4, Ireland
[18]Department of Diabetes, Faculty of Life Sciences and Medicine, King's College London, London, UK
[19]Florence Nightingale Faculty of Nursing, Midwifery & Palliative Care, King's College London, London, UK
[20]Department of Medical Psychology, Radboud University Medical Center, Nijmegen, The Netherlands
[21]Diabetes Research Centre, University of Leicester, Leicester, UK

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CONFLICT OF INTEREST STATEMENT

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author, Prof. Dr. Julia K. Mader, upon reasonable request.

ORCID

Monika Cigler https://orcid.org/0000-0001-5827-7991
Omaima El-Hakouni https://orcid.org/0009-0009-1616-6711
Renald Mecani https://orcid.org/0000-0003-2697-8544

François Pouwer https://orcid.org/0000-0002-8172-9818
Evertine J. Abbink https://orcid.org/0009-0006-5424-3704
Michaela Moser https://orcid.org/0009-0009-7249-797X
Bastiaan E. de Galan https://orcid.org/0000-0002-1255-7741
Eric Renard https://orcid.org/0000-0002-3407-7263
Mark Evans https://orcid.org/0000-0001-8122-8987
Julie Maria Bøggild Brøsen https://orcid.org/0000-0001-7361-4407
Ulrik Pedersen-Bjergaard https://orcid.org/0000-0003-0588-4880
Rory J. McCrimmon https://orcid.org/0000-0002-3957-1981
Simon Heller https://orcid.org/0000-0002-2425-9565
Petra M. Baumann https://orcid.org/0000-0002-7665-0929
Uffe Søholm https://orcid.org/0000-0003-0848-9421
Silvia Basta https://orcid.org/0009-0008-5234-7547
Patrick Divilly https://orcid.org/0000-0001-6916-3164
Natalie Zaremba https://orcid.org/0000-0002-1720-1621
Stephanie A. Amiel https://orcid.org/0000-0003-2686-5531
Pratik Choudhary https://orcid.org/0000-0001-7635-4735
Julia K. Mader https://orcid.org/0000-0001-7854-4233

REFERENCES

1. Lind M, Polonsky W, Hirsch IB, et al. Design and methods of a randomized trial of continuous glucose monitoring in persons with type 1 diabetes with impaired Glycaemic control treated with multiple daily insulin injections (GOLD study). *J Diabetes Sci Technol*. 2016;10(3):754-761. doi:10.1177/1932296816642578
2. Beck RW, RiddLesworth T, Ruedy K, et al. Effect of continuous glucose monitoring on Glycaemic control in adults with type 1 diabetes using insulin injections: the DIAMOND randomized clinical trial. *JAMA*. 2017;317(4):371-378. doi:10.1001/jama.2016.19975
3. Aleppo G, Ruedy KJ, RiddLesworth TD, et al. REPLACE-BG: A randomized trial comparing continuous glucose monitoring with and without routine blood glucose monitoring in adults with well-controlled type 1 diabetes. *Diabetes Care*. 2017;40(4):538-545. doi:10.2337/dc16-2482
4. Leelarathna L, Evans ML, Neupane S, et al. Intermittently scanned continuous glucose monitoring for type 1 diabetes. *N Engl J Med*. 2022;387(16):1477-1487. doi:10.1056/NEJMoa2205650
5. Haak T, Hanaire H, Ajjan R, Hermanns N, Riveline JP, Rayman G. Flash glucose-sensing technology as a replacement for blood glucose monitoring for the Management of Insulin-Treated Type 2 diabetes: a multicenter, open-label randomized controlled trial. *Diabetes Ther*. 2017;8(1):55-73. doi:10.1007/s13300-016-0223-6
6. Bolinder J, Antuna R, Geelhoed-Duijvestijn P, Kroger J, Weitgasser R. Novel glucose-sensing technology and hypoglycaemia in type 1 diabetes: a multicentre, non-masked, randomised controlled trial. *Lancet*. 2016;388(10057):2254-2263. doi:10.1016/S0140-6736(16)31535-5
7. RiddLesworth T, Price D, Cohen N, Beck RW. Hypoglycaemic episode frequency and the effect of continuous glucose monitoring in adults with type 1 diabetes using multiple daily insulin injections. *Diabetes Ther*. 2017;8(4):947-951. doi:10.1007/s13300-017-0281-4
8. Garg SK, Hirsch IB, Repetto E, et al. Impact of continuous glucose monitoring on hospitalizations and glucose control in people with type 2 diabetes: real-world analysis. *Diabetes Obes Metab*. 2024;26(11):5202-5210. doi:10.1111/dom.15866
9. Sherr JL, Laffel LM, Liu J, et al. Severe Hypoglycaemia and impaired awareness of Hypoglycaemia persist in people with type 1 diabetes despite use of diabetes technology: results from a cross-sectional survey. *Diabetes Care*. 2024;47(6):941-947. doi:10.2337/dc23-1765
10. International Hypoglycaemia Study Group. Glucose concentrations of less than 3.0 mmol/L (54 mg/dL) should Be reported in clinical trials: A joint position statement of the American Diabetes Association and the European Association for the Study of diabetes. *Diabetes Care*. 2017;40(1):155-157. doi:10.2337/dc16-2215 Epub 2016 Nov 21. PMID: 27872155.
11. Divilly P, Zaremba N, Mahmoudi Z, et al. Hypo-METRICS: Hypoglycaemia-MEasurement, ThResholds and ImpaCtS-A multi-country clinical study to define the optimal threshold and duration of sensor-detected hypoglycaemia that impact the experience of hypoglycaemia, quality of life and health economic outcomes: the study protocol. *Diabet Med*. 2022;39(9):e14892. doi:10.1111/dme.14892
12. de Galan BE, McCrimmon RJ, Ibberson M, et al. Reducing the burden of hypoglycaemia in people with diabetes through increased understanding: design of the Hypoglycaemia REdefining SOLutions for better liVEs (hypo-RESOLVE) project. *Diabet Med*. 2020;37(6):1066-1073. doi:10.1111/dme.14240
13. Divilly P, Martine-Edith G, Zaremba N, et al. Relationship between sensor-detected hypoglycaemia and patient-reported hypoglycaemia in people with type 1 and insulin-treated type 2 diabetes: the hypo-METRICS study. *Diabetes Care*. 2024;47(20):1769-1777. doi:10.2337/dc23-2332
14. Mainz J, Kristensen S, Roe D. The power of the patient's voice in the modern health care system. *International J Qual Health Care*. 2022;34(1):ii1-ii2. doi:10.1093/intqhc/mzac001
15. Gold AE, MacLeod KM, Frier BM. Frequency of severe hypoglycaemia in patients with type I diabetes with impaired awareness of hypoglycaemia. *Diabetes Care*. 1994;17(7):697-703. doi:10.2337/diacare.17.7.697
16. Danne T, Nimri R, Battelino T, et al. International consensus on use of continuous glucose monitoring. *Diabetes Care*. 2017;40(12):1631-1640. doi:10.2337/dc17-1600
17. Zammitt NN, Streftaris G, Gibson GJ, Deary IJ, Frier BM. Modeling the consistency of hypoglycaemic symptoms: high variability in diabetes. *Diabetes Technol Ther*. 2011;13(5):571-578. doi:10.1089/dia.2010.0207
18. Amiel SA, Sherwin RS, Simonson DC, Tamborlane WV. Effect of intensive insulin therapy on Glycaemic thresholds for

Counterregulatory hormone release. *Diabetes*. 1988;37(7):901-907. doi:10.2337/diab.37.7.901
19. Boyle PJ, Schwartz NS, Shah SD, Clutter WE, Cryer PE. Plasma glucose concentrations at the onset of hypoglycaemic symptoms in patients with poorly controlled diabetes and in nondiabetics. *N Engl J Med*. 1988;318(9):1487-1492. doi:10.1056/NEJM198806093182302
20. Mizoguchi T, Aoyama N, Jinnouchi Y, Inoue M, Eguchi E, Ohira T. Associations of fluctuations in blood glucose and insulin with hypoglycemic symptoms. *Sci Rep*. 2025;15:11579. doi:10.1038/s41598-025-91544-5
21. Weber KK, Lohmann T, Busch K, Donati-Hirsch I, Riel R. High frequency of unrecognized hypoglycaemias in patients with type 2 diabetes is discovered by continuous glucose monitoring. *Exp Clin Endocrinol Diabetes*. 2007;115(8):491-494. doi:10.1055/s-2007-984452
22. Martine-Edith G, Zaremba N, Divilly P, et al. Associations between Hypoglycaemia awareness status and symptoms of Hypoglycaemia among adults with type 1 or insulin-treated type 2 diabetes using the hypo-METRICS smartphone application. *Diabetes Technol Ther*. 2024;26(8):566-574. doi:10.1089/dia.2023.0596
23. Søholm U, BroadLey M, Zaremba N, et al. The impact of hypoglycaemia on daily functioning among adults with diabetes: a prospective observational study using the hypo-METRICS app. *Diabetologia*. 2024;67(10):2160-2174. doi:10.1007/s00125-024-06233-1
24. Søholm U, BroadLey MM, Zaremba N, et al. Impact of nocturnal hypoglycaemia on daily sleep quality, mood, and alertness among adults with type 1 diabetes: the hypo-METRICS (Hypoglycaemia measurement, thresholds, and Impacts) study. *Diabetes*. 2022;71(Suppl 1):233. doi:10.2337/db22-233-OR
25. Mahmoudi Z, Del Favero S, Jacob P, Choudhary P. Toward an optimal definition of Hypoglycaemia with continuous glucose monitoring. *Comput Methods Programs Biomed*. 2021;209:106303. doi:10.1016/j.cmpb.2021.106303
26. Gonder-Frederick LA, Zrebiec JF, Bauchowitz AU, et al. Cognitive function is disrupted by both hypo- and hyperglycaemia in school-aged children with type 1 diabetes: a field study. *Diabetes Care*. 2009;32(6):1001-1006. doi:10.2337/dc08-1722
27. Heller SR, Cryer PE. Reduced neuroendocrine and symptomatic responses to subsequent hypoglycaemia after 1 episode of hypoglycaemia in nondiabetic humans. *Diabetes*. 1991;40(2):223-226. doi:10.2337/diab.40.2.223
28. Davis MR, Mellman M, Shamoon H. Further defects in counterregulatory responses induced by recurrent hypoglycaemia in IDDM. *Diabetes*. 1992;41(10):1335-1340. doi:10.2337/diab.41.10.1335
29. Verhulst CEM, Fabricius TW, Nefs G, et al. Consistent effects of hypoglycemia on cognitive function in people with or without diabetes. *Diabetes Care*. 2022;45:2103-2110. doi:10.2337/dc21-2502
30. Mellor J, Kuznetsov D, Heller S, et al. Estimating risk of consequences following hypoglycaemia exposure using the hypo-RESOLVE cohort: a secondary analysis of pooled data from insulin clinical trials. *Diabetologia*. 2024;67:2210-2224. doi:10.1007/s00125-024-06225-1

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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