Response to the Letter to the Editor regarding Lipoedema – myths and facts, Part 1 and Part 5. European Best Practice of Lipoedema – Summary of the European Lipoedema Forum consensus. Phlebologie 2020; 49: 31–49

We are delighted to receive the letter from the USA; it shows that our series of articles is not only read in Germany and Europe but worldwide. Two and a half years after the publication of part 1, all 5 articles of the series can be found among the top 10 "most read articles" of the "Phlebologie" (1, August 2020) [1]. In Europe as well, the "Myths and Facts about Lipedema" have received a great deal of attention and contribute further to a much-needed paradigm shift regarding this disease. Renowned Lipedema experts from 10 European countries support the consensus on lipedema elaborated in part 5 of the series [2]. In "Pathways", the official magazine of the Canadian Lymphedema Framework, the myths about lipedema as well as the paradigm shift will be the main topic of the autumn issue.

We are aware that in the USA the old narrative regarding lipedema is still being retained. There, three patient organizations with strong media coverage and strong financial backing as well as a few physicians closely associated with these organizations dominate the opinion on lipedema: Lipedema Simplified by Catherine Seo [3], Fat Disorder Research Society (FDRS) [4] and Lipedema Foundation [5], the latter two being interconnected closely. Felicitie Daftuar is both a member of the leadership team of FDRS [6] and founder and Executive Director of Lipedema Foundation [7]. This is important to note, as the author of the letter to the editor – Karen Herbst – is very closely connected to FDRS [8] as well as to Lipedema Foundation [9]. Since 2015 Felicitie Daftuar has been supporting Karen Herbst's projects with millions of Dollars [10, 11].

Before we discuss the co-signers of the letter to the editor, we will clarify the disputed points A to F.

The author of the letter begins her letter with the statement: *„Lipedema is a disease of loose connective tissue (LCT) – not just fat. Constituents of LCT include cells and an extracellular matrix of collagen fibers around glycosaminoglycans (GAGs) bound to sodium and water*."

However, there is no scientific evidence of lipedema being a disease of the loose connective tissue; the author does not provide any scientific reference supporting her statement. In contrast, the statement of the loose connective tissue having an extracellular matrix that consists of fibers and basic substance (here also glycosaminoglycans) is correct. It is also true that glycosaminoglycans have a high water-binding capacity. The connection between glycosaminoglycans (GAGs) and lipedema is explained by the author in point A.

Regarding A. Edema

She writes "*Edema is defined as excess interstitial fluid (IF) (2) that is free, or bound in a GAG gel as in lipedema.*"

While the first half sentence of this statement certainly represents correct and general medical knowledge, the second half sentence ("or bound in GAG gel as in lipedema") lacks any reliable proof. The source for this statement is the author's own work mentioned under A.2, which will be discussed further below.

Regarding 1.

The described "overgrowth of blood vessels in lipedema tissue" was only found in the obese lipedema study population of the mentioned study of Al-Ghadban, not in the group of non-obese [12]. Thus, the described changes are much more likely to be attributed to obesity and not to the lipedema syndrome. In the recently published work by Felmerer, Hägerling and Gousopoulos in pure lipedema patients, only "increased adipocyte size" could be detected. Although the VIPAR method of 3-D reconstruction developed by Hägerling was used in this histopathological examination, there was no evidence of altered lymphatic or blood vessels in lipedema patients [13].

The co-author Gousopoulos gave a presentation on the results of the work mentioned by the letter at the German Congress of Lymphology 2019 in Bad Krozingen. His conclusion, which can also be read in the congress abstract, is: "No morphological changes in the lymphatic component are present in lipedema" [14]. Furthermore, there is no scientific evidence of "leaky capillaries" in the lipedema syndrome. The source given by the author dates in 1986 and did not discuss the lipedema syndrome.

Regarding 2.–6.

Points 2 to 6 regularly refer to the concept of existing GAGs in lipedema. The only source mentioned to describe this concept of GAGs in lipedema is a work by the author of the letter herself. For those interested in Herbst's understanding of science, this paper ("Lipedema Is Not Just Fat") is recommended [15]. The statements she made there were in no way comprehensible to us. Neither do the sources mentioned by Herbst give any indication as to how GAGs are connected to lipedema. It is true that the review by Reed and Rubin mentioned by Herbst deals in detail with glycosaminoglycans – but in a completely different context. GAGs do not occur in the studies mentioned by the author of the letter, and the references to lipedema seem to us – with all due respect – to be far-fetched. The question what the postulated GAGs have to do with the strong pain symptoms reported by patients remains unsolved.

It is therefore not surprising that this concept has not been published in a scientific (peer reviewed) journal, but on her own website Lipedema.com [16], which she operates together with co-signatories and lipedema activists (Kahn, Iker and Ehrlich) [17]. Herbst and the same co-signatories are also the editors of a book (Lymphedema and Lipedema Nutrition Guide: Foods, Vitamins, Minerals and Supplements), for which the chairman of the Lipedema Foundation, Felicitie Daftuar, wrote the foreword [18].

Yes, we know this may read a bit complicated, but it is important to understand how "science" works in the US when financially strong patient organizations – with their own interests – enter close cooperation with practitioners.

Regarding the facts

Edema are defined as "abnormal accumulation of fluids" in the tissue [19]. However, neither clinical examination nor imaging could ever prove a relevant edema in patients with lipedema syndrome [20]. A multi-centric study using high resolution ultrasound on patients with the diagnosis lipedema did not find any proof of fluids within the soft tissue of the legs [21]. In a study published in 2020, in which patients with lipedema syndrome are examined by MR lymphography, the authors conclude: "The fat tissue was homogenous, without any signs of edema in pure lipedema patients" [22]. The presence of edema has never been described in histological examinations either [20]. Reich-Schupke, Altmeyer and Stücker wrote in their pioneering article in 2012: "The term 'lipedema' is actually misleading, since it is not an edema in a sense of accumulation of fluid in the tissue" [23]. This was confirmed by the authors of the Dutch lipedema guideline, in which they described lipedema as an "unfortunate term", as it suggests fluid in the tissue where no fluid can be found [24]. Finally, the European Lipedema Forum – an international group of high-ranking experts from seven European countries – summarized in a highly recognized consensus paper: "There is **no** scientific evidence that Lipedema is an 'edema problem'" (emphasized in the original) [25].

Regarding B: Manual Lymph therapy and compression

Besides their own pilot study on 7 (!) patients treated with "whole body manual subcutaneous adipose tissue (SAT) therapy", the only therapy studies focusing on lipedema mentioned in this section by the author of the letter are the two studies by Szolnoky et al. The scientists investigated the effect of Complete Decongestive Therapy (CDT) on lipedema. CDT consists of manual lymphatic drainage (MLD), compression, movement and exercise therapy, skin care (recently also with additive self-management) [26]. Patients in this study reported an improvement of symptoms under CDT.

Unfortunately, the author of the letter to the editor confuses MLD and CDT. The study of Szolnoky et al. does not clarify which of the above-mentioned components of CDT have actually been effective. Compression and sports are effective in the treatment of lipedema due to their anti-inflammatory effects. This was described in part 5 of the article series and is consistent with the European Consensus [25]. The effectiveness of MLD, as claimed by the American author, remains without evidence.

Furthermore, the question arises: If, as explained above, there is no evidence of relevant fluids in lipedema, what is the point of the repeatedly demanded drainage? Which substance in the soft tissue of the legs should be drained with MLD, fatty tissue?

Regarding C: Lipohypertrophy

The painless and complaint-free increase of leg volume is called lipohypertrophy in German speaking countries, in accordance with Herpertz [27]. This is a far-reaching consensus and makes sense. If the disproportionality of the legs – which is perceived subjectively by the patient – would already fulfill the criteria for the diagnosis of lipedema, the door would be opened to the abuse of any kind of therapy (especially regarding the current ideal of beauty, which prefers thin legs and is propagated in online-media). The author of the letter to the editor further writes: "*A woman with painful lipedema who underwent therapy and has no pain, still has lipedema*". We agree with this statement. Similarly, a patient with arterial hypertension who has normal blood pressure after taking antihypertensive medication, still has arterial hypertension. However, before taking the drugs, the physician measured elevated blood pressure. The same applies to the symptoms of lipedema syndrome.

Regarding D: Obesity, secondary lymphedema and bariatric surgery

Obviously, lipedema, lymphedema and obesity are quite different diseases. In order to differentiate these, the experienced clinician does not need a biomarker. However, over 80 % of patients diagnosed with lipedema are obese, about 50 % even morbidly obese, which means they have a BMI ≥ 40 kg/m² [28–33]. On these facts, the international data is very consistent. In other words: lipedema and obesity occur together in the vast majority of women. Morbid obesity can lead to a lymphatic drainage disorder or lymphedema in some patients. The pathophysiology of this is well documented and – usually – not disputed [34, 35]. Patients with obesity-associated lymphedema are the fastest growing group of patients in the European Center for Lymphology in Hinterzarten.

The author of the letter to the editor writes: "Many severe obese women with lipedema do not develop lymphedema". We agree with this statement. However, not every "chain smoker" suffers from bronchial carcinoma. And certainly, the author agrees with us that "chain smoking" carries a significantly increased risk of developing bronchial carcinoma.

We are glad that the author mentions the biomarker PF 4 which is currently propagated by Stanley Rockson and is increasingly being promoted in lipedema patient forums. According to Rockson et al., PF 4 hints to a lymphatic dysfunction underlying lipedema: "Finally, our results support the prevailing hypothesis, that in lipedema, lymphatic dysfunction plays a role" [36]. In a current webinar of Lipedema Simplified (the other above-mentioned lipedema patient organizations), Rockson presents a patient – supposedly – suffering from lipedema ("This patient has lipedema"), showing that PF 4 was increased [37]. The here presented lipedema patient obviously suffers from a (probably obesity-associated) lymphedema with already existing stasis dermatosis! We can believe that PF 4 is increased in those patients. However, the correct diagnosis of the patients is essential to enable the production of valid and serious results. This also

applies to imaging studies. Already earlier clinical studies using indirect lymphography and lymph scintigraphy showed that lymph transport from the subepidermal compartment is not impaired in lipedema patients [38–40]. Furthermore, recent studies with modern histopathological examination methods did not provide evidence of altered lymphatic vessels in lipedema patients [13].

The data on long-term success of bariatric surgery in morbid obesity is very consistent and convincing [41–49]. A current study of the University Hospital in Freiburg in cooperation with the European Center for Lymphology in Hinterzarten regarding the effects of bariatric surgery in severely obese patients with lipedema syndrome shows a success of this therapeutic option and confirms our experiences made since 2008 with this group of patients [50].

Regarding E: Psychology

Here, too, we disagree with the author of the letter to the editor. In a study of 150 patients with a confirmed diagnosis of lipedema syndrome, 80 % of the women presented mental disorders (mostly depression, eating disorders or post-traumatic stress disorder) or severe psychological impairment such as burnout or chronic stress. However, these disorders or severe mental impairments existed before the development of lipedema-associated pain in soft tissue [29]. Purely in terms of formal logic – and this has been verified – something that temporally precedes the development of lipoedema cannot be its consequence. In other words: the lipedema syndrome cannot be the cause of this psychological stress. The influence of psychological stress and mental disorders on the development of pain as well as on pain perception has been studied extensively and is very consistent [51–57]. Compared to the 12-month prevalence of these mental disorders in the general population [58], depression and eating disorders were significantly increased in the 12-month period before the onset of lipedema-associated pain symptoms [29]. Therefore, the statement "psychological factors can contribute significantly to the development of lipedema", which was criticized by the author of the letter, is well documented.

Regarding F: Liposuction

We know that many patients disagree on performing liposuction depending on the body weight (and therefore the Body Mass Index).

The European Consensus, which was criticized by the author of the letter, considers a BMI of 35 kg/m² with simultaneous central obesity as the upper limit for liposuction. Above a BMI of 35 kg/m² (with rare exceptions), the obesity of the lipedema patient is in the foreground and should be focused on. The European Consensus recommends reviewing the indication for bariatric surgery. This corresponds to the recommendations of the S3 guidelines of the German Society of Surgery of Obesity. The positive effect of bariatric surgery on the lipedema syndrome has been investigated and confirmed [50]. We consider liposuction in patients with clear central obesity to be malpractice. The criteria for liposuction were clearly defined in the European Consensus [2].

In Germany, too, the – largely similar – assessment of the Federal Joint Committee (GBA) led to an outcry of indignation from patient representatives. The self-help associations described the GBA's decision as a "farce" [59]. In a press release worth reading (because it is demasking) issued by the organised self-help group for women with lipedema on 9-20-2019, liposuction is hyped as a "saving treatment", "to prevent a life in a wheelchair" or even a "very premature mortality" [60]. Nothing could be further from reality! Neither does pure lipedema (without being accompanied by severe obesity) lead to a "life in a wheelchair" nor to a "strongly premature mortality". Furthermore, we are not aware of any data that indicate liposuction to be a "saving treatment" regarding the prognoses presented above.

However, the reasoning of the author of the letter is also noteworthy: "We disagree that Liposuction is not a treatment option in patients with a BMI > 35 kg/m² and central obesity (WHtR > 0.5) [1] Women can develop lipedema LCT on the abdomen [14]". As a source for this "lipedema of the abdomen", the author again cites one of her own publications.

The lipedema of the abdomen was already mentioned in a critical letter to the editor by Schmeller in 2018. Thus, Schmeller wrote: "The association of subcutaneous fat increases localized at trunk and extremities as consequence of obesity or as consequence of the lipedema ... can be very difficult, partly probably also impossible". And further down: "Whether the adipocytes of the lipedema cause an increase in volume and weight viscerally in the abdominal area in addition to the subcutis of the extremities can currently not be assessed with certainty" [61].

In the following, we will quote our replica to Schmeller's thoughts at the time [62]. "We would like to emphasize that subcutaneous fat tissue proliferations on the abdomen or adipocytes in the abdominal cavity should NEVER be brought into a connection with lipedema! Thought games such as this one lack any scientific evidence. In these regions there are no pain symptoms reported by the patients. However, we can already see – on a daily basis – patients who are presented to us with a diagnosis of lipedema (or "lipolymphedema") and who are convinced that their lipedema is the cause of their weight gain, their obesity. More and more often we see obese patients who claim to suffer from "whole-body lipedema" or "lipedema of the abdomen" – which is also often certified by medical colleagues – and who see this as the cause of their weight gain. Here we would like to shout out loud to the author of the letter: "HOLD MR. SCHMELLER, NO; PLEASE DO NOT OPEN THIS PANDORA'S BOX!" (end of quote). It seems that the box has now been opened.

Ultimately, this letter to the editor also reflects the controversial debates of recent years, which the first author of the series of articles with Karen Herbst, but also with Felicitie Daftuar, has held on stages and conferences in the past. We are concerned that the very narrow US-American view of lipedema, a view that ignores the problem of obesity as well as the – pre-existing – psychological burdens on women, does not do justice to the complexity of this disease.

A final word on the co-signers of this letter. Nearly every person listed there is connected with "The US Standard of Care Committee". If you enter this supposed organization in Google, you will not find any entry. However, you can find them on Herbst's own website "lipedema.com" mentioned above, where the author of

the letter, Karen Herbst, was also able to publish her contribution to the GAGs. There, they refer to a meeting that took place in April 2019 in Baltimore and in which 18 of the 22 co-signers took part. The author of the letter to the editor and the two patient organisations FDRS and the Lipedema Foundation mentioned above [63] had invited to this conference. One of the aims of the meeting in Baltimore was to develop US guidelines on lipedema. Imagine this situation in Germany (or other European countries): Lipedema self-help groups organize a conference, invite "self-selected physicians" and then develop a so-called guideline.

Research and therapy that is independent of interests, which would be essential especially in the case of lipedema, certainly works differently.

Further, other co-signatories who do not belong to this group also have close links to the Lipedema Foundation [64, 65].

CONCLUSION

Money makes the world go round – this also applies to patient-funded and thus controlled research into lipedema in the USA.

Conflict of Interest

The authors declare that they have no conflict of interest.

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References

[1] Abrufbar unter: https://www.thieme-connect.com/products/ejournals/topten/10.1055/s-00034913
[2] Bertsch T, Erbacher G, Corda D et al. Lipoedema – myths and facts, Part 5. European Best Practice of Lipoedema – Summary of the European Lipoedema Forum consensus. Phlebologie 2020; 49: 31–49
[3] Abrufbar unter: https://lipedema-simplified.org
[4] Abrufbar unter: https://www.fatdisorders.org
[5] Abrufbar unter: https://www.lipedema.org
[6] Abrufbar unter: https://www.fatdisorders.org/leadership-team
[7] Abrufbar unter: https://www.lipedema.org/about/#anchor-who
[8] Abrufbar unter: https://globalgenes.org/event/fdrs-2020-focus-on-fat-disorders/
[9] Abrufbar unter: https://www.lipedema.org/biobank-imaging-patient-treat-uofa-arizona-herbst-research-grant
[10] Abrufbar unter: http://www.curelipedema.org/treat
[11] Abrufbar unter: https://www.lipedema.org/patient-centered-research
[12] AL-Ghadban S, Cromer W, Allen M et al. Dilated Blood and Lymphatic Microvessels, Angiogenesis, Increased Macrophages, and Adipocyte Hypertrophy in Lipedema Thigh Skin and Fat Tissue. Journal of Obesity 2019; 2019: 4–10
[13] Felmerer G, Hägerling R, Gousopoulos E et al. Adipose Tissue Hypertrophy, An Aberrant Biochemical Profile and Distinct Gene Expression in Lipedema. J Surg Res 2020; 253: 294–303
[14] Gousopoulos E. Absence of lymphatic morphological changes but a distinct biochemical niche underlie lipedema development. Vortrag auf der Lymphologie 2019 in Bad Krozingen. Abstractband S. 21
[15] Abrufbar unter: https://www.lipedema.com/lipedema-is-not-just-fat
[16] Abrufbar unter: https://www.lipedema.com/us-lipedema-soc/#join
[17] Abrufbar unter: https://www.lipedema.com/about
[18] https://www.amazon.de/Lymphedema-Lipedema-Nutrition-Guide-supplements/dp/0976480689
[19] Abrufbar unter: https://www.pschyrembel.de/Ödem/K0FKR
[20] Bertsch T, Erbacher G. Lipödem – Mythen und Fakten Teil 2. Phlebologie 2018; 47: 120–126
[21] Hirsch T, Schleinitz J, Faerber G et al. Is the differential diagnosis of lipoedema by means of high-resolution ultrasonography possible? Phlebologie 2018; 47 (4): 182–187
[22] Cellina M, Gibelli D, Soresina M et al. Non-contrast MR Lymphography of lipedema. Magent Resonance Imaging 2020; 71: 115–124
[23] Reich-Schupke S, Altmeyer P, Stücker M. Thick legs – not always lipedema. J Dtsch Dermatol Ges 2013; 11 (3): 225–233
[24] Lipedema – Guidelines in the Netherlands 2014. Abrufbar unter https://www.gdlymph.eu/assets/pdf/Dutch-lipoedema-guideline-2014.pdf
[25] Bertsch T, Erbacher G, Corda D et al. Lipoedema – myths and facts, Part 5. European Best Practice of Lipoedema – Summary of the European Lipoedema Forum consensus. Phlebologie 2020; 49: 31–49
[26] Abrufbar unter: https://www.awmf.org/leitlinien/detail/ll/058-001.html
[27] Herpertz U. Das Lipödem. Lymphologie 1995; 19: 1–7
[28] Child AH, Gordon KD, Sharpe P et al. Lipedema: An inherited condition. Am J Med Genet Part A 2010; 152A: 970–976
[29] Erbacher G, Bertsch T. Lipoedema and pain: what is the role of the psyche? Results of a pilot study with 150 lipedema patients. Phlebologie 2020; 49: 305–316. doi:10.1055/a-1238-6657
[30] Dudek JE, Białaszek W, Ostaszewski P et al. Depression and appearance-related distress in functioning with lipedema. Psychology, health & medicine 2018; 23 (7): 846–853
[31] Dudek JE, Bialaszek W, Ostaszewski P. Quality of life in women with lipoedema: a contextual behavioral approach. Quality of Life Research 2016; 25: 401–408
[32] Bosman J. Lipoedema: Poor knowledge, neglect or disinterest? Journal of Lymphoedema 2011; 6 (2): 109–111
[33] Bertsch T, Erbacher G. Lipödem – Mythen und Fakten Teil 3. Phlebologie 2018; 47: 188–197
[34] Bertsch T. Adipositas-assoziierte Lymphödeme – unterschätzt und unterbehandelt. Phlebologie 2018; 47: 75–83
[35] Mehrara B, Greene A. Lymphedema and Obesity: Is There a Link? Plast Reconstr Surg 2014; 134 (1): 154–160
[36] Ma W, Gil HJ, Escobedo N et al. Platelet factor 4 is a biomarker for lymphatic-promoted disorders. JCI Insight 2020; 5 (13): e135109. Published 2020 Jul 9. doi:10.1172/jci.insight.135109
[37] Lipedema simplified, webinar: Abrufbar unter. https://lipedema-simplified.org/webinar2020-pf4/ Hier Minute 14:37 bis 14:47. Videosequenz kann aber auch beim Erstautor (Bertsch) angefragt werden
[38] Harwood CA, Bull RH, Evan J et al. Lymphatic and venous function in lipoedema. Br J Dermatol 1996; 134 (1): 1–6
[39] Bräutigam P, Földi E, Schaiper T et al. Analysis of lymphatic drainage in various forms of leg edema using two compartment lymphoscintigraphy. Lymphology 1998; 31 (2): 43–55
[40] Amann-Vesti BT. Druckmessung in den initialen Lymphgefäßen der Haut bei Patienten mit Lipödem. LymphForsch 2002; 6 (1): 7–9
[41] Buchwald H, Estok R, Fahrbach K et al. Weight and type 2 diabetes after bariatric surgery: systematic review and meta-analysis. Am J Med 2009; 122: 248–256

[42] Wittgrove AC, Clark GW. Laparoscopic Gastric Bypass, Roux en-Y – 500 Patients: Technique and Results, with 3–60 month follow-up. OBES SURG 2000; 10: 233
[43] Sugerman HJ et al. Diabetes and hypertension in severe obesity and effects of gastric bypass-induced weight loss. Ann Surg 2003; 237: 751–756
[44] Rasheid S, Banasiak M, Lipska A et al. Bypass is an Effective Treatment for Obstructive Sleep Apnea in Patients with Clinically Significant Obesity. OBES SURG 2003; 13: 5
[45] Courcoulas AP, Yanovski S, Arterburn DE et al. Long-term Outcomes of Bariatric Surgery: A National Institutes of Health Symposium. JAMA Surg 2014; 149 (12): 1323–1329
[46] Arterburn DE, Olsen M, Maciejewski M et al. Association Between Bariatric Surgery and Long-term Survival. JAMA 2015; 313 (1): 62–70
[47] Sjöström L, Nabro K, Sjöström D et al. Effects of bariatric surgery on mortality in Swedish obese subjects. N Engl J Med 2007; 357: 741–752
[48] Adams TD, Gress R, Hunt S et al. Long-term mortality after gastric bypass surgery. N Engl J Med 2007; 357: 753–761
[49] Shubeck S, Dimmick J, Telem D. Long-term Outcomes Following Bariatric Surgery. JAMA 2018; 319 (3): 302–303
[50] Fink JM, Schreiner L, Bertsch T et al. Leg Volume in Patients with Lipoedema following Bariatric Surgery. Visc Med 2020. doi:10.1159/000511044
[51] Linsmayer D, Neidlinger PK, Braus DF. Rheuma und Psyche – Eine Kurzübersicht. Orthopade 2019; 48 (11): 957–962. doi:10.1007/s00132-019-03812-8
[52] Baerwald C, Manger B, Hueber A. Depression als Komorbidität bei rheumatoider Arthritis. Z Rheumatol 2019; 78: 243–248 https://doi.org/10.1007/s00393-018-0568-5
[53] Hofmann P, Hemberger S, Lunzer R et al. Psychische Aspekte der rheumatoiden Arthritis Wie ansprechen? Wie behandeln? Zusammenfassung des Expertenmeetings „Rheuma trifft Psyche“ am 05.11.2015. Abrufbar unter: ttps://www.pfizermed.at/sites/default/files/rheuma_trifft_psyche.pdf
[54] Bischoff N, Morina N, Egloff N. Chronischer Schmerz bei Traumatisierung. Komplexität und Herausforderung bei Diagnostik und Therapie. PiD – Psychotherapie im Dialog 2016; 17 (4): 69–72. doi:10.1055/s-0042-116706
[55] Viana MC, Lim CCW, Pereira FG et al. Prior mental disorders and subsequent onset of chronic back or neck pain: findings from 19 countries. J Pain 2018; 19 (1): 99–110. doi:10.1016/j.jpain.2017.08.011
[56] Tegethoff M, Belardi A, Stalujanis E et al. Comorbidity of Mental Disorders and Chronic Pain: Chronology of Onset in Adolescents of a National Representative Cohort. The Journal of Pain 2015; 16 (10): 1054–1064. doi:https://doi.org/10.1016/j.jpain.2015.06.009
[57] Hooten WM. Chronic Pain and Mental Health Disorders. 2016 Mayo Foundation for Medical Education and Research, Mayo Clin Proc 2016; 91(7): 955-970. Abrufbar unter: https://www.mayoclinicproceedings.org/article/S0025-6196(16)30182-3/pdf
[58] Jacobi F, Höfler M, Siegert J et al. Twelve-month prevalence, comorbidity and correlates of mental disorders in Germany: The Mental Health Module of the German Health Interview and Examination Survey for Adults (DEGS1-MH). Int J Methods Psychiatr Res 2014; 23 (3): 304–319
[59] Deutsches Ärzteblatt Dienstag, 24. September 2019. Abrufbar unter: https://www.aerzteblatt.de/nachrichten/106211/Betroffene-enttaeuscht-ueber-G-BA-Entscheidung-zur-Liposuktion
[60] Gemeinsame Presseerklärung der organisierten Selbsthilfe von Frauen mit Lipödem vom 20.9.2019. Abrufbar unter: https://webcache.googleusercontent.com/search?q=cache:gnx6Dnkwr1sJ:https://www.lipoedem-fakten.de/app/download/6021579766/Gemeinsame%2BPresseerkl%25C3%25A4rung%2BLip%25C3%25B6dembetroffener%2Bvom%2B20.09.2019.pdf%3Ft%3D1579150244+&cd=1&hl=de&ct=clnk&gl=de&client=safari
[61] Schmeller W. Letter to the Editor: Zu den drei Beiträgen: T. Bertsch und G. Erbacher: Lipödem – Mythen und Fakten Teil 1 bis 3. Phlebologie 2018; 47: 376–379
[62] Bertsch T, Erbacher G. Stellungnahme der Autoren auf Letter to the Editor von Schmeller W. Phlebologie 2018; 47: 379–384
[63] Abrufbar unter: https://www.lipedema.com/us-lipedema-soc/#supporters
[64] Abrufbar unter: https://www.lipedema.org/namri-mri-crescenzi-donahue-research-grant
[65] https://www.lipedema.org/hyaluronan-extracellular-matrix-harten-research-grant/

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