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Research Article

The effects of sex and gender role on responses to pressure pain

Die Auswirkungen des biologischen Geschlechts und der Geschlechterrolle auf Druckschmerz

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Abstract

Background: Several studies on experimental mechanical pain suggested a strong influence of sex demonstrating females to be more sensitive. We examined the hypothesis that not only sex but also gender role affects pain responsiveness and looked for mediators of this effect.
Method: As indicators of pain the threshold the intensity and the unpleasantness of pressure stimuli were measured, as well as sensory and affective quality of pain. The gender role of 74 students was assessed by the Bem Sex Role Inventory (BSRI). Furthermore several psychological variables assumed to be potential mediators (catastrophising, fear of pain, depressive symptoms, pain coping) were obtained.
Results: ANOVA revealed significant main effects of sex in all pain variables except affective quality of pain. Contrary to our hypothesis gender role had no influence on pain responses, neither was there an interaction of sex and gender. Fear of pain just missed the significance level identifying it as mediator of the sex effect on affective pain.
Conclusions: In summary, our study corroborated previous findings that women are more responsive to mechanical pain stimuli with effect sizes being medium to large, whereas gender role did not predict any of the assessed pain parameters. No convincing evidence was found that the influence of sex is predominantly mediated by psychological characteristics of the individual.

Keywords: gender role, sex, pain responsiveness, fear of pain

Zusammenfassung

Hintergrund: Eine Reihe von Studien zur experimentellen Schmerzwahrnehmung (Druckschmerz) zeigte, dass weibliche Versuchsteilnehmer sensitiver auf Schmerzreize reagierten. Wir untersuchten die Hypothese, dass nicht nur das biologische Geschlecht, sondern auch die Geschlechterrolle die Schmerzreaktivität beeinflussen und ob Mediatoren dieser Effekte zu identifizieren sind.
Methode: Als Schmerzreaktionsparameter wurden die Schmerzschwelle, die Intensität und Unangenehmheit der Schmerzreize erhoben, ebenso wie die sensorische und affektive Schmerzqualität. Die Geschlechterrolle von 74 Pbdn. wurde über das Bem Sex Role Inventory erfasst. Verschiedene psychologische Variablen, von denen angenommen wurde, dass sie eine Mediatorfunktion haben könnten, wie die Katastrophisierung, Schmerzangst, depressive Symptomatik und Schmerzbewältigungsstrategien wurden ebenfalls erhoben.
Ergebnisse: ANOVAs zeigten signifikante Haupteffekte des Faktors biologisches Geschlecht bei allen Schmerzvariablen mit der Ausnahme der affektiven Schmerzqualität. Entgegen unserer Hypothese hatte die Geschlechterrolle keinen Einfluss auf die Schmerzreaktionen, noch ergab sich ein Interaktionseffekt. Angst vor Schmerz verfehlte das für die Feststellung einer Mediation festgesetzte Signifikanzniveau knapp.

Schlussfolgerungen: Die Ergebnisse bestätigten die bisherigen Befunde einer erhöhten Schmerzsensibilität von Frauen hinsichtlich mechanischer Reize, wobei sich mittlere bzw. hohe Effektstärken ergaben. Dagegen konnte die Geschlechterrolle keine Varianz aufklären. Es wurde zudem keine überzeugende Evidenz dafür gefunden, dass psychologische Variablen (habituelle Eigenschaften der Person) den Einfluss des biologischen Geschlechts mediieren.

Schlüsselwörter: Geschlechterrolle, biologisches Geschlecht, Schmerzwahrnehmung, Schmerzangst

Introduction

It is well known that the prevalence of chronic musculoskeletal pain is higher in women than in men [1]. Furthermore, most studies on laboratory pain demonstrated that women report higher pain intensity, especially regarding mechanical stimuli [2], [3], and demonstrate lower pain thresholds [4], [5], [6], [7]. With respect to other modalities of pain stimuli, findings are more controversial [8], [9], [10], [11].

The mechanisms of the enhanced sensitivity to mechanical stimuli in women are not yet fully understood. However, the influence of gonadal hormones has been empirically substantiated [12].

Hypotheses associating the differences between sexes with psychosocial factors have also been advanced [13]. The concept of gender roles assumes that a female or male identity is mainly determined by cultural and social norms or differential reinforcement of behaviour. Thus, the behaviour of expressing one's pain could be shaped by social norms and reinforcement. A plausible assumption is that females willingly reveal their pain and receive positive social feedback, whereas males are not encouraged or even punished for expressing their pain [14]. Some studies supported the influence of role stereotypes on pain. For instance, male participants revealed less pain when being tested by female experimenters wishing presumably to appear as a "tough guy" in front of an attractive woman [15]. Therefore, we would expect individuals endowed with a distinct feminine role concept to express more pain.

Only a few studies so far have examined the relation between gender role and experimental pain and reported some controversial results. Otto and Dougher [7] found that in men masculinity correlated with the mechanical pain threshold level. Myers et al. [16] showed gender role identity to be associated with pain tolerance, but not with the pain threshold. Sanford et al. [17], furthermore, reported femininity, but not masculinity to correlate with pain tolerance. On the contrary, Fillingim et al. [18] did not find any influence of gender role on thermal pain sensitivity in women.

We intended to re-examine the effect of sex and gender on pain sensitivity to mechanical stimuli in a laboratory environment. Gender role was conceptualised in line with Bem's conception of androgyny (BSRI; [19]). According to Bem [19], it is defined by the two independent dimensions of "masculinity" and "femininity". Hence, females and males can have a more female or, respectively, male self-concept according to the difference between the values on the feminine and the masculine scale. We assumed that gender role predicts pain responsiveness in addition to sex. An interaction between both factors was also expected.

Furthermore, we wanted to examine whether psychological trait characteristics act as a mediator between sex or gender and pain. As potential mediators we selected the following variables because there was evidence that they correlate to pain: *depressive symptoms* (DE: [20], [21], [22]), *catastrophising* (CA: [23], [24], [25]), *fear of pain* (FoP: [26], [27], [28]) and *pain coping strategies* (CO: [29], [30]). They were reported to also differ between females and males (DE: [31]; CA: [32], CO: [33]; FoP: [34]).

Various pain parameters (threshold (PT), intensity (PI), unpleasantness (PU), sensory and affective quality of pain (SP, AP)) were assessed. The following assumptions were examined:

- Both gender role and sex and their interaction explain differences in pain responsiveness (two main effects and an interaction effect in a two-factorial analysis of variance).
- The selected psychological variables correlate with sex and gender as well as pain and are, assumingly, mediators of the effects of sex and/or gender.

Methods

Sample

Subjects were students recruited within the University. They were informed by notices of a study on pressure pain perception and that they would either receive credits for their participation, or a small monetary reward. Before the experiment informed consent was obtained from each subject. The study was approved by an institutional review board. Their mother tongue had to be German to prevent misunderstandings regarding the questionnaires that needed to be filled in. Exclusion criteria were acute pain at the time of the experiment and/or persistent pain, consummation of alcohol on the day of the experiment and pain medication during the 48 hours preceding the experiment. Pregnancy prohibited participation as well as bruises or injuries at the site of the pain application

(forearm). We recruited 35 women and 39 men with a mean age of 23.1 years (SD=2.5; range 18–33).

Design

Independent organismic variables were sex and gender role. Dependent variables were PT, PI and PU, as well as SP and AP. Potential mediators of the effects of sex or gender (DE, CA, FoP, COP) were assessed by questionnaires. As a control variable, blood pressure, which is known to influence pain responses and can differ between sexes [35], was measured as well as the menstrual phase and the intake of hormonal contraceptives in women.

Sex and gender role identity as independent variables

Subjects documented their sex in a personal questionnaire and filled in the German version of the BSRI [19], [36]. The subject had to rate each of the 60 items of the inventory (adjectives) on a 7-point scale regarding how well it describes the person's self-concept. "Masculine" items are, for example, "strong-willed", "competitive" and "vigorous". "Feminine" items are "romantic", "sensitive" and "cheerful". The so-called "androgyny score" was determined by calculating the difference of means of the two scale scores (femininity/masculinity) and dividing it by the standard deviation of these difference scores. These androgyny scores were normally distributed in our study. Negative scores represent an androgyny score with more masculine attributes, and positive scores represent a predominantly feminine gender role.

Parameters of pain responsiveness

The stimuli were applied by means of a Fischer pressure algometer [37]. The intensity was regulated by the experimenters, who pressed a force gauge fitted with a rubber tip (1 cm^2) as steadily as possible onto the skin of the subject. The gauge's rubber tip was placed on the inner forearm between wrist and arm crook at distance of 4–5 cm from the skin fold near the elbow.

After some practice, a good reliability of the procedure can be expected (see [38]; ICC=0.80–0.92). Pre-tests had revealed a good reliability (re-test and inter-experimenter) of threshold measures (pre-test: interval of trials 5 minutes; four trials, two experimenters).

Before measuring the pain responses in the main study, subjects received three test stimuli on the right and left thigh to acquaint them with the procedure. The two experimenters were female.

Pain threshold

The experimenters increased the pressure from 0 kg by 1 kg/sec in a continuous manner up to the individual PT indicated by the subject loudly saying "Now!". The value displayed on algometer's panel at this moment was documented. This procedure was repeated 5 times with an interval of 1 minute, the mean score was used. The sites of application were alternated (left/right and vice versa). The site of the first stimulus was determined randomly.

Pain intensity and unpleasantness

After an interval of 3 minutes a standard pressure stimulus set at 5.5 kg and held for 5 seconds was used to determine PI and PU. This stimulus was expected to be perceived as relatively painful (4–5 on a NRS=0–10; personal communication by Lautenbacher; pretests). The ratings of PI and PU were performed with an 11-point numerical rating scale [0 (no pain) to 10 (greatest imaginable pain); 0 (not unpleasant at all) to 10 (extremely unpleasant)]. The procedure was repeated 3 times on each arm (interval 1 minute, site of first trial chosen randomly). Scales were presented to the subjects directly after each trial.

Pain quality

After the application of the standard stimulus within the six trials the questionnaire for sensory and affective pain quality (Schmerzempfindungsskala; SES; [39]) was presented to the subjects. The questionnaire measured the sensory dimension (SP) of pain sensation by 10 items, like "pulsating" and "stinging", and the affective dimension (AP) by 14 items, like "intolerable" or "exhausting" on a 4-point scale. A sum score was calculated for each scale. Reliability of the scales is reported to be high (Cronbach's α=.81–.96).

Potential mediator variables

All questionnaires assessing the mediator variables and the BSRI were filled in after the experiment. **CA** (13 items, 5-point rating scale) was assessed by the German version of the Pain Catastrophising Scale adapted by Crombez et al. [40] based on the test constructed by Sullivan et al. [23]. It was translated back and forth by English/German native speakers. Only the total score was used in our study. Homogeneity of the three subscales was reported to be adequate (α=.68–.87; [41]).

Fear of pain (**FoP-F**) was assessed the by the **F**ear of Pain Questionnaire III (FPQ-III: 30 items; [41]) in the German version (translation procedure, see above). Reliability scores within the range of .80–.83 were found. The Instrument requires subjects to indicate on a 5-point scale (1: not at all to 5: very much) how much fear they feel, when imagining situations in which three types of pain ("strong", "weak" or "medical") are experienced. Situations are e.g. "*having received an injection in the mouth*" (medical pain), "*biting one's tongue*" (weak pain) or "*breaking an arm*" (strong pain).

Since fear of pain (**FoP-P**) is conceptualized quite differently by the often used short form of the Pain Anxiety Symptoms Scale (PASS; [42]), it was also applied in the

German version [43]. Typical items are <*When I sense pain, I feel dizzy or faint*> and <*I try to avoid activities when I am hurt*>. Homogeneity of scales is reported to be good (α=.75–.91; see [44]). The total score was used. To assess coping we applied the Pain Coping Questionnaire (PCQ, 39 items; 5-point-scale) by Reid et al. [45]. It distinguishes between "approach" (CO-a; e.g. information seeking) "emotion focussed avoidance" (CO-efa; e.g. worrying) and "problem focussed avoidance" (CO-pfa; e.g. positive self-instructions). Cronbach's α of these scales was reported to vary between .85 and .89.

DE was measured with the Beck Depression Inventory (BDI [46]; German version [47]), as the internationally most frequently applied inventory for the assessment of affective dysfunctions. Internal consistency varies between .74 and .92 [47].

Control variables

Systolic and diastolic **blood pressure** were obtained by a calibrated automatic standard tourniquet technique (Sanitas, Type SBM 12) applied to the left upper arm, after subjects had filled in the questionnaires and relaxed for 2 minutes.

Menstrual phase was assessed by determining the time interval between the experiment and the first day of the last menstruation. Subjects were categorised as being in the "follicular phase" (day 1–8), the "ovulatory phase" (day 9–17) or the "luteal phase" (day 18–28). Also, the use of hormonal contraceptives was documented (yes/no).

Statistical analysis

Power analysis was conducted before the experiment. Assuming a small to moderate effect size (0.45), a sample size of n=54 is needed when requiring a beta of .80 and an alpha of 0.5% in analysis of variance. Hypotheses regarding the expected main and interaction effects were tested by analysis of variance, and mediator analysis was guided by the suggestions made by Baron and Kenny [48]. The level of statistical significance was set at α=0.05.

Results

Preliminary analyses

Control variables were correlated to pain response variables to determine whether they should be used as covariates in the analysis of variance. A significant ($p<0.05$) correlation of systolic blood pressure was found regarding pain intensity ($r=-0.28$) and unpleasantness ($r=-0.26$). Menstrual phase of female subjects showed no association to pain responses, nor did the use of hormonal contraceptives (F-scores <0.1, $p>0.05$). Also, the interaction terms did not reach significance (all $p>0.05$).

Androgyny scores were normally distributed with M=0.089 (SD=0.875). ANOVA with sex as group variable and either feminine or masculine scale scores resulted in non-significant F-values ($F<1$). Androgyny scores were divided at the median (0.126) yielding a binary variable (gender role more feminine (M=0.794) / gender role more masculine (M=–0.616)).This procedure allowed the use of ANOVA for testing the main hypotheses.

Hypothesis testing

The main analyses of pain response parameters were carried out by 2x2 of the androgyny scores (BSRI). In the analyses of PI and PU systolic blood pressure was entered as a covariate. Significant differences between male and female subjects were observed in both variables (see Table 1 and Table 2), but not between feminine and masculine subjects. Regarding all other pain variables only a main effect of sex appeared. PI, PU and SP were higher in females whereas PT was lower. AP only showed a trend towards significance. Post-hoc tests documented an interaction effect regarding the variable SP: it was highest in masculine women, while masculine men scored lowest.

As a last step in the analysis, it was examined whether the psychological variables could be viewed as mediators of sex. Since no gender effects were found, a comparable analysis regarding gender was not indicated (see Table 2, using gender as a metric predictor led to similar results which were omitted for frugality). The prerequisite for a variable to act as a mediator is that it has to correlate with the dependent variables (c, see Figure 1), in this case the different pain parameters, and show a significant a path (correlation to the predictor variable, see Figure 1). Four variables reached the significance level regarding the correlation to sex (see Table 3, row A). With the exception of DE and CO-efa, all other variables showed significant correlations with at least some of the pain measures. FoP-P showed the strongest associations. A mediator effect is corroborated when there is distinct difference between c and c' Figure 1). An effect like this appeared regarding FoP-P and FoP-F, in terms of the pain measures AP and SP (Table 4). However, the Sobel test clearly denied significance of the indirect effect of sex on SP mediated by FoP-F. Regarding FoP-P the significance level was just exceeded ($p=0.054$).

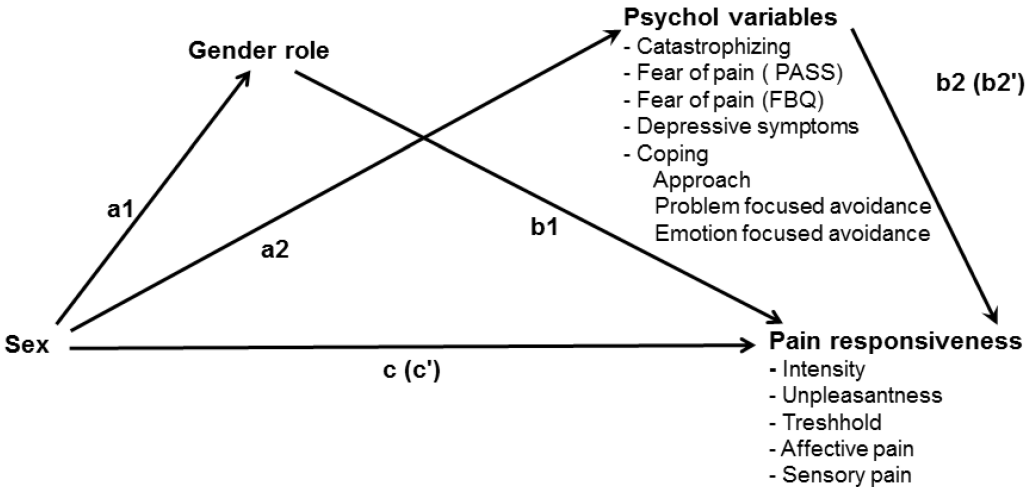

Figure 1: Model of associations between sex, gender role, psychological factors (mediators) and pain parameters (a–c: standardized beta coeff; c', b': beta weights in the last step of mediator analysis only performed for sex as independent variable)

Table 1: Means and standard deviations of pain responses and psychological variables

Gender	**Sex female (n=39)**			**Sex male (n=35)**
Feminine (n=37)	**Pain var.**	Mean	Sd	Mean
	PI	7.03	1.35	5.19
	PU	6.76	1.26	4.95
	PT	3.04	1.30	3.72
	SP	18.24	4.67	17.55
	AP	24.18	9.27	22.65
	Psychol. var.			
	DE	8.35	4.15	6.80
	CA	18.65	9.35	17.30
	FoP-P	34.59	14.45	30.30
	FoP-F	80.76	16.23	71.50
	CO-a	3.24	0.52	3.12
	CO-efa	2.09	0.57	1.78
	CO-pfa	3.07	0.67	3.15
Masculine (n=37)	**Pain var.**			
	PI	6.42	1.82	5.42
	PU	6.57	1.60	4.91
	PT	2.86	0.78	3.99
	SP	20.67	5.21	15.42
	AP	25.56	7.69	20.79
	Psychol. var.			
	DE	5.94	4.92	4.79
	CA	19.17	6.29	17.0
	FoP-P	39.44	11.34	30.32
	FoP-F	81.28	12.33	75.47
	CO-a	3.44	0.48	3.06
	CO-efa.	2.09	0.67	1.81
	CO-pfa	2.86	0.62	2.91

PI pain intensity; **PU** pain unpleasantness; **PT** pain threshold; **SP** sensory pain; **AP** affective pain;
DE depression; **CA** catastrophising; **FoP-P** Fear of Pain (PASS); **FoP-F** Fear of Pain (FBQ);
CO-a coping: approach; **CO-efa** Coping: emotion focused avoidance;
CO-pfa coping: problem focused avoidance

Table 2: Results of 2x2 analyses of variance of pain variables

Variable	F (df=1.70)	p
pain intensity (PI)*		
Sex	6.01	**0.017***
Gender	0.40	0.529
S x G	1.08	0.302
pain unpleasantness (PU)*		
Sex	12.56	**0.001***
Gender	0.10	0.756
S x G	0.04	0.85
pain threshold (PT)		
Sex	12.96	**0.001***
Gender	0.03	0.871
S x G	0.83	0.365
sensory pain (SP)		
Sex	7.43	**0.008***
Gender	0.02	0.889
S x G	4.39	**0.04***
affective pain (AP)		
Sex	3.09	0.083
Gender	0.02	0.893
S x G	0.82	0.369

* p<0.05

Table 3: Correlation matrix including all pain response parameters and psychological variables

var.	Sex	PI	PU	PT	AP	SP	D	C	FoP-F	FoP-P	co-a	co-efa
Sex												
PI	**.38****											
PU	**.46****	**.86****										
PT	**–.39****	**–.33****	**–.35****									
AP	.20	**.44****	**.46****	**–.31****								
SP	**.30****	.25*	.27*	–.17	**.71****							
D	.14	.11	.05	.05	.17	.18						
C	.12	.27*	**.30 ****	–.02	**.38****	**.32***	.28*					
FoP-F	.24*	**.30****	.26*	–.14	**.32****	.28*	.15	**.37****				
FoP-P	.25*	**.38****	**.43****	–.09	**.49****	**.51****	.28*	**.79****	**.48****			
co-a	.26*	.09	.14	.08	.15	.23*	.04	**.31****	**.36****	**.36****		
co-efa	.25*	.14	.11	–.01	.14	.20	**.34****	**.55****	**34****	**.55****	**.35****	
co-pfa	–.06	.28*	–.28*	.22	–.07	–.07	–.06	–.19	–.10	–.19	.09	–.08

PI pain intensity; **PU** pain unpleasantness; **PT** pain threshold; **SP** sensory pain; **AP** affective pain; **DE** depression; **CA** catastrophising; **FoP-P** (PASS); **FoP-F** (FBQ); **CO-a** coping: approach; **CO-efa**. Coping: emotion focused avoidance; **CO-pfa** coping: problem focused avoidance
* p≤0.05 ** p≤0.01

Table 4: Mediator analysis (only conducted in case of beta coefficients **b2** and **a2** being significant)

pain variable / c	psychological variable	c'	b2'	Sobel Test
AP / .20*	**FoP-P**	.09 ns	.46 **	p=0.054
SP / .30**	**FoP-F**	.19 ns	*.46***	p=0.131

AP affective pain; **SP** sensory pain; **FoP- P** (PASS); **FoP-F** (FBQ);
a2, b2, b2', c, c', see Figure 1
*p<0.05 **p<0.01

Discussion

Sex and gender role as moderators of pain

A very clear result of our study is that females respond in a more sensitive manner to mechanical pain stimuli than males. They exhibited a lower pain threshold, described the pressure stimulus (lying in the medium range of intensity) as being more intense and unpleasant and used a greater number of descriptors for the sensory characteristics of the stimuli. Though affective pain did not differ significantly between sexes, females tended to evaluate the pressure stimuli as emotionally more aversive. Effect sizes range from d=.41 (AF) to d=1.04 (PI). In this respect, the findings of the majority of earlier studies were replicated (e.g. [2], [49]) and extended by additional data on pain parameters so far rarely assessed in the context of gender studies.

This study was based on a sample of subjects in their early adulthood (year of birth 1974–1982), i.e. on individuals from different birth cohorts than those reported on in earlier publications (see [49]: year of birth of participants in 16 studies: 1904–1972). Thus, it can be concluded that sex differences in response to mechanical pain are not birth cohort dependent. Compared to effect sizes reported by Riley et al. [49] for experimental pain (d=.56) those observed in our study are even higher (average d=.77). In their latest review Fillingim et al. [3] underline that the higher pain responsivity in females is one of the most stable findings in clinical and experimental pain research.

The expected effect of *gender role identity* analysed as a dichotomous variable, however, was not observed in any of the analyses; the association with pain response parameters (as indicated by explained variance in ANOVA) was close to zero. Moreover, no interaction of sex and gender was observed. Hence gender role did not explain any variance in pain responses above that which was explained by sex. Furthermore our additional expectation of gender role to be a mediator of sex effects was not confirmed.

Our results are thus in accordance with those found by Fillingim et al. [18], who also came to a negative conclusion regarding the effect of gender role. The results of three studies which had demonstrated some effect of gender on some pain response parameters – Otto and Dougher [7], Myers et al. [16] and Sanford et al. [17] – had been inconclusive in regard to the direction and the domains of influence. The recent review on gender role effects given by Fillingim et al. [3] in his extensive overview on sex and gender differences in pain supports the inconsistency of findings in this area of research.

Psychological variables as potential mediators

Fear of pain as measured by PASS was closely associated with most pain variables (exception PU). FBQ scores correlated only with PI and AP. Thus, it seems that the PASS catches the cognitive-emotional characteristics of fear and its influence on the processing of pain, especially concerning its experienced quality, more adequately than the FBQ.

Only with regard to the response variable PT, we saw no variance at all explained by the psychological characteristics of the individual. Though it is generally suggested that the pain threshold is less sensitive to psychosocial factors, in two studies an association with anxiety or depressive symptoms had been observed [26], [50].

Also, pain *catastrophising* showed significant correlations to most pain variables, thus confirming the findings of other studies on *chronic* [51], [52], [53], [54] and *laboratory* pain [55], [56].

Against expectations (see [33]), coping did not correlate significantly with pain. Only the coping strategy "problem-focussed avoidance" came close to significance regarding PI and PU. It has to be pointed out that – contrary to the usual labelling – avoidance as defined here denotes an active effort of pain control and seems to be a more or less functional strategy. In summary, different coping strategies had a very limited effect on pain experience.

In contrast to other studies [31], [57], [58], [59] depressive symptoms, though being somewhat higher in females then in males, did not significantly differ between sexes, which was also observed in regard to catastrophising. The failure to find sex differences may be blamed on the overall low level of depression and catastrophising found in our student sample and on the level of androgyny in the sample with the average androgyny score close to zero.

Having to discard the hypothesis of gender role being a mediator of sex effects, we examined the importance of our mediator variables in terms of biological sex. The analysis according to the Baron and Kenny [48] suggested two mediators, i.e. FoP-P and FoP-F. Both, however, failed to meet the statistical criterion when a Sobel test was applied. Fear of Pain as defined by the PASS missed it very curtly.

Exploratory findings

The control variables menstrual phase and intake of hormonal contraceptive had no influence on the pain response of female subjects. As they are only crude indicators of the hormonal state, hormonal influences on pain cannot be ruled out by our study. It was confirmed that blood pressure is negatively associated with pain intensity, thus corroborating the assumptions of the central inhibitory effect of blood pressure via baroreceptor activity [57]. It was also strongly correlated to sex, with females showing a lower systolic blood pressure. Although

only planned post-hoc, we also examined the possible mediator function of blood pressure with reference to sex but found no evidence for it.

Limitations of the study

The instrument employed to determine gender role identity was developed in the late 1980s (German version). It might have lost some validity regarding the description of femininity and masculinity and thus failed to grasp the current gender role differences. About twenty publications on the BSRI were found from 1990 until today, mostly demonstrating plausible findings regarding the validity of the androgyny concept including two recent studies [60], [61]. Thus, there is no reason to generally doubt the validity of the instrument.

However, the distribution of femininity and masculinity in our study is probably not representative of German young adults, since the participants of our study were university students and predominantly androgynous the two biological sexes not even differing from another regarding femininity and masculinity. For this reason, the study should be extended to samples of greater diversity of gender attributes to validate the presented conclusions. Moreover, it can be questioned whether the sex of the experimenters exerted some influence on the results, as it is known that this can have an effect on the pain response [61]. The differences in pain responsiveness between both sexes in our sample may have been increased by the fact that the experimenters were female. There is, however, no plausible assumption that this abolished an effect of gender. Systematically varying the sex of the experimenters would have overstrained the experiment with a 2x2x2 factorial design, doubling the number of participants needed. Nevertheless, this limits the generalisability of the results to a certain extent.

Also it has to be admitted that the large number of ANOVAs (5) and correlations calculated aimed at the identification of mediators also weakens generalisability.

Summery and conclusions

Summarising the results, we find no evidence that gender role, as assessed by the BSRI, exerted any influence on pain responses, whereas sex did. Fear of pain measured by PASS, being higher in women, came close to being a mediator between sex and pain. Sex, however, remained an independent predictor regarding pain in all other analyses. Consequently, psychological differences between the sexes, as far as they were examined in our study, did not explain the sex differences found in pain responsiveness. The variable (blood pressure) we examined post-hoc as a potential biological mediator also did not contribute to the explanation.

Hence the mechanisms that underlie the corroborated sex differences in pain responsiveness to pressure stimulation are far from being fully understood and there is much inconsistency in regard to findings on the significance of gender. As lately emphasised by Greenspan et al. [62] much more sophisticated and interdisciplinary research is needed in this field.

List of abbreviations

AP affective pain
BSRI Bem Sex Role Inventory
CA catastrophising
CO-a coping: approach
CO-efa coping: emotion focused avoidance
CO-pfa coping: problem focused avoidance
DE depression
FBQ Fear of Pain Questionnaire
FoP-P fear of pain measured by PASS
FoP-F fear of pain measured by FPQ
PASS Pain Anxiety Symptoms Scale
PI pain intensity
PU pain unpleasantness
PT pain threshold
SP sensory pain

Notes

Competing interests

The authors declare that they have no competing interests.

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Please cite as

Kröner-Herwig B, Gaßmann J, Tromsdorf M, Zahrend E. The effects of sex and gender role on responses to pressure pain . GMS Psychosoc Med. 2012;9:Doc01.
DOI: 10.3205/psm000079, URN: urn:nbn:de:0183-psm0000792

This article is freely available from

http://www.egms.de/en/journals/psm/2012-9/psm000079.shtml

Published: *2012-02-28*

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