Thermal signatures of human pheromones in sexual and reproductive behaviour

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Chemically mediated sexual communication in humans has been largely neglected due to its non-conscious and relatively concealed nature. However, menstrual cycle synchronisation, puberty onset in young pre-pubertal girls exposed to their stepfather, and consanguinity avoidance suggest a function in the physiological regulation of sexual and reproductive behaviour in humans. These phenomena are related to activation of the limbic system by pheromones. Based on sexually dimorphic activation of brain hypothalamic areas and the control of body temperature via the hypothalamus, our hypothesis is that human sexual pheromones can induce thermal effects that can be revealed by high-resolution thermal infrared imaging. Here we show that in women, male sexual pheromones induce thermal effects that are linked to the ovarian cycle. These findings suggest a dramatic influence of pheromones on human sexual and reproductive behaviour through neuroendocrine brain control, established on the plesiomorphic nature of chemical communication across species.

Pheromones are striking species-specific chemosignals that control a wide range of social behaviours in animals (Karlson and Luscher, 1959). These are otherwise unimportant chemical compounds that range in structure from volatile small molecules

to peptides and steroids, and they have been selected through evolution and combined in individual-specific concentrations within species (Wyatt, 2009). In both animals and humans, the pheromonal identity of any given individual has a decisive role in the sexual and reproductive behaviour relating to their genetics identification, providing avoidance of consanguinity (Roberts and Little, 2008). However, pheromone-mediated sexual communication in humans has not been investigated comprehensively, generally because of its non-conscious, concealed nature and the neglected functions of the vomeronasal organ (Jacob et al, 2001b; Monti-Bloch et al., 1998; Meredith, 2001; Wysocki and Preti, 2004). Indeed, it is commonly believed that pheromonal communication in mammals belongs to a unique and specialised pathway, the vomeronasal system, and that the main olfactory epithelium is responsible exclusively for sensing common odours (Keverne, 2004).

More recently, however, a new perspective in pheromone perception has emerged following the discovery of a new class of evolutionarily preserved chemoreceptors, the trace amine-associated receptors, that are involved in the detection of pheromonal cues (Liberles and Buck, 2006). These findings support the involvement in the human chemo-sexual communication of a pathway that is mediated via the main olfactory system (Shepherd, 2006; Savic et al, 2009).

Despite these considerations, in human, pheromones can elicit sexually dimorphic activation of hypothalamus–amygdale areas, independent of whether they are sensed through the vomeronasal system or the main olfactory system, or both (Savic et al, 2000; Savic et al, 2001; Savic and Lindström, 2008; Brancucci et al, 2008). This resembles a plesiomorphic plan of the mammalian pheromonal perception pathway, as has been shown in rodents, which show sexually dimorphic neuronal activation at multiple levels (Segovia and Guillamon, 1996; Keverne, 2004; Dean et al., 2004). Moreover, pheromones can regulate the physiology and behaviour of animals and

humans, as seen by menstrual cycle synchronisation and the Vandenberg effect, which involves the female control of sperm competition and the onset of puberty (McClintock, 1971; Ellis and Garber, 2000). These findings reveal the evolutionary roots and the chemical nature of sexual function in humans.

Of particular interest, human pheromones activate the hypothalamus (Swaab et al, 2001; Savic et al, 2001; Wyart et al, 2007), which controls the autonomic nervous system (ANS), a key structure in sexual arousal, sex-hormone release, reproductive behaviour, and body-temperature regulation. Thus, autonomic parameters can vary in humans exposed to pheromones (Monti-Bloch et al, 1998; Bensafi et al, 2004a, b; Jacob et al., 2001b). In particular, androstadienone (4,16-androstadien-3-one), a sex-steroid-derived compound, can induce dose-dependent and gender-specific non-conscious effects on ANS responses and on peripheral physiological activity and mood (Jacob et al., 2001b; Bensafi et al, 2004a,b). A noticeable effect here is the variation of hedonic perception across the ovarian cycle that is associated with another putative human pheromone, androstenone (5-α-androst-16en-3α-on), which elicits neutral or unpleasant smell sensations according to the ovulatory and non-ovulatory periods, respectively (Hummel et al 1991; Grammer, 1993). Similarly, the preferences of women for male faces, voices, masculinity and behavioural displays, and the neuromodulation of the reward system depend on the phases of the ovarian cycle (Dreher et al, 2007; Jones et al, 2008; Gangestad and Thornhill, 2008). Although odorant perception and olfactory thresholds have been associated with the ovarian cycle and the hormone status, nothing has been specifically established with respect to pheromone perception and its relation to the ovarian cycle and hormone status (Navarrete-Palacios et al, 2003; Doty and Cameron, 2009). It has also been demonstrated that ovarian hormones can control sexual arousal and that women present a sexual arousal peak around ovulation (Adams et al, 1978; Matteo and Rissman, 1984; Wallen, 1990).

Therefore, even 50 years after their discovery, the open question remains whether sexual and reproductive behaviour in humans depends on interactions between these sexual pheromones and the hormone status. To investigate this question, we compared the dynamics of facial cutaneous temperature in women during their ovulatory versus non-ovulatory conditions, while they were exposed to androstadienone. Indeed, facial cutaneous temperature is controlled by the ANS, and it might thus represent a fundamental communication tool for sexual arousal (Prause and Heiman, 2009; Kukkonen et al., 2009).

We used here high-resolution thermal infrared imaging to monitor the patterns and time-courses of facial temperature (Pavlidis et al, 2002; Merla and Romani, 2007; Shastri et al, 2009). The other ANS parameters that were recorded included pulse rate, breathing rate, galvanic skin response, and palm temperature, as relevant system-response controls.

These signals were recorded across three experimental phases: adaptation, during which time the women adapted to the experimental set-up while being exposed to the placebo (double-distilled water); stimulation, with the women exposed to androstadienone; and recovery, with the women again exposed to the placebo. In the control experiments, the placebo was instead administered during all three of these phases. Baseline normalisation and trend corrections were then applied to analyse the time-courses of the temperature curves.

Figure 1 shows representative high-resolution images of the facial temperature changes induced during placebo exposure and exposure to the pheromone androstadienone in ovulatory and non-ovulatory women under the adaptation (AP), stimulation (SP) and recovery (RP) periods. These temperature modifications showed region-based amplitude variations, within a range of 28 °C to 37 °C.

The exposure of the ovulatory and non-ovulatory women to the pheromone produced marked differences in both the spatial patterns (Fig. 1) and time-courses (Fig. 2) of the whole-face temperatures across the experimental phases. For these average group temperatures, during the stimulation and recovery periods the whole-face temperatures increased with the ovulatory women and decreased with the non-ovulatory women (Fig. 2). Indeed, for androstadienone exposure as recovery period vs. stimulation period, there was a significant continuous increase in the ovulatory women ($p < 0.001$; paired t-test) and a significant continuous decrease in the non-ovulatory women ($p < 0.05$; paired t-test), compared to placebo exposure, where no changes were seen. This group analysis thus highlighted the dramatic influence of the ovulatory condition on the temperature time-course induced by exposure to the pheromone.

When pixel-by-pixel subtraction algorithms were applied to these thermal image series (after movement correction and image re-alignment), the representative high-resolution subtraction image series shown in Figure 3 highlighted the temperature differences in specific facial regions across the ovulatory conditions and time-course phases. In particular, with the ovulatory women, androstadienone exposure induced scattered-shaped increases in the temperatures for the nose, forehead, lachrymal gland and peri-oral region. Conversely, androstadienone administration with the non-ovulatory women caused scattered decreases in the temperatures in these same regions. The control (placebo) exposure produced only a generalised cooling of the face temperature, with this placebo trend adjusted for in the following data analysis.

These findings prompted us to further investigate the time-courses in these specific localised facial regions, which correspond to responsive areas for neurophysiological activation described in the literature (Pavlidis, 2002; Shastri, 2009). Figure 4 shows the group-averaged temperatures across the full time-courses for the pheromone (and placebo) exposure periods for six specific regions of interest (ROIs):

the right and left lachrymal regions and cutaneous projections of the corrugator muscles, and the forehead and the nose. The temperatures across these ROIs showed essentially similar behaviours. In the ovulatory women, exposure to androstadienone initially showed little change from placebo during the exposure (stimulation) period, except for a significant activation peak more specifically for the right lachrymal region and the nose ($p < 0.05$ and $p < 0.01$, respectively). Then upon removal of androstadienone, there were rises in the temperatures across these ROIs for the duration of the recovery period. In contrast, in the non-ovulatory women, exposed to androstadienone, there was an almost immediate cooling down of all of these facial regions (except for the nose) that generally continued through the recovery period. Paired t-tests for phase-to-phase comparisons highlighted the significant differences in the temperature variations across these ROIs, which were not seen under placebo administration or during the adaptation phase (Table 1).

The largest variations here were seen for the nose, the temperature of which appeared to show a significant late activation peak during the recovery phase following androstadienone exposure in both the ovulatory and non-ovulatory women ($p < 0.001$ for both), as also seen for the forehead region with the non-ovulatory women (Fig. 4).

Figure 5 shows the time variations of the group-averaged autonomic parameters recorded, following baseline normalisation and placebo-trend correction. Here, the palm temperature reproduced the nose and forehead temperature time-courses, thus suggesting a systemic effect of androstadienone on temperature control. Moreover, in the ovulatory women, the palm temperature appeared to be the most responsive parameter to androstadienone administration. Similarly, the galvanic skin response showed a dramatic increase in the ovulatory women during the recovery phase.

In particular, the pulse rate also significantly increased with androstadienone exposure under both ovulatory and non-ovulatory conditions (Fig. 4). These were seen as higher values with the ovulatory women during the exposure, although during the recovery phase, the pulse rates of the non-ovulatory women increased such that they matched those of the ovulatory women at the end of the observation period (Fig. 4). Finally, the breathing rates significantly decreased with androstadienone administration under both the ovulatory and non-ovulatory conditions, with a larger decrease seen during the androstadienone exposure for the non-ovulatory women. Again, during the recovery phase after androstadienone exposure, the breathing rates of the ovulatory and non-ovulatory women reached similar final values, while remaining significantly lower than their control values.

In summary, the androstadienone administration appears to activate different ANS responses according to the physiological, ovulatory versus non-ovulatory, conditions of these women. These ANS responses appear to resemble antagonistic parasympathetic–sympathetic-like effects, which might be responsible for the scattered or oscillatory behaviour found on both the spatial and time patterns of these temperature changes. Thus, both of these systems respond to androstadienone, but to different levels that depend on the physiological status of the woman.

The sex-specific effects of androstadienone on the ANS and on mood that are reported in the literature are generally not very consistent across studies. Indeed, both sympathetic-like and parasympathetic-like effects have been reported in women (Jacob et al., 2001a; Grosser et al., 2000). Furthermore, the time-courses of the responses to exposure to androstadienone range across studies, from about 6 min, to 30-40 min, and up to more than 2 h (Jacob and McClintock, 2000; Grosser et al., 2000; Bensafi et al., 2003). These discrepancies might reflect the various methodological approaches and the dose-dependent effects of the ANS responses that are seen between the sexes: at higher

concentrations, androstadienone has sympathetic-like effects in women, and parasympathetic-like effects in men (Bensafi et al., 2003; Bensafi et al., 2004b). Incongruities across these studies can also be ascribed to pheromone solvent and stimulus interactions, or to sexual differences (Wysocki et al, 2009). However, these data can be explained by the action of two hormones, as androgens and oestrogens, that separately stimulate antagonistic parasympathetic and sympathetic systems, resulting in synergistic effects on libido and sexual arousal at different levels in both men and women.

Such sexual arousability is related to a natural asymmetric balance of hormones that can induce 'lopsided' sympatho-parasympathetic effects. This asymmetric balance of hormones is the more functional relationship between the two autonomic components, which act agonistically on the parasympathetic and sympathetic centres (Motofei and Rowland, 2005). At the same time, none of these previous studies have shown differences between women and across their physiological/ hormone status. These different responses in women might be related to the hormonal cycle being ultimately responsible for changes in facial appearance (Law Smith et al, 2006), mood (Jacob et al., 2001a; Bensafi et al, 2004a,b) and sexual arousal (Matteo and Rissman, 1984), and for neurobiological and neurofunctional modulation of the reward systems (Dreher et al, 2007). Furthermore, the increased availability, receptivity and desire that can occur during a woman's ovulatory period might have arisen from the evolutionary pressure to facilitate procreation.

Different techniques and methodological approaches have established that androstadienone elicits mood alterations, pulsatile secretion of luteinising hormone, high level of cortisol, specific brain-region activation, and ANS responses (Grosser et al., 2000; Savic et al., 2001; Jacob et al., 2001a, 2002; Preti et al, 2003, Bensafi et al., 2003, 2004; Wyart et al, 2007; Savic and Lindström, 2008). In particular, electrodermal

activity is considered a responsive indicator of autonomic sympathetic arousal (Critchley, 2002; Bensafi et al., 2002), while skin temperature has been reported as a parasympathetic indicator (Jacob et al., 2001a, b). Finally, measures such as heart rate and respiration activity are considered more variable in these studies (Bensafi et al., 2004b). However, wide discrepancies in the autonomic nervous responses subsequent to androstadienone exposure have been reported as effects that appear not to be related to physiological status (Grosser et al., 2000; Jacob et al., 2001a; Bensafi et al., 2004b).

One aspect of our data shows changes in the rates and time-courses of the autonomic parameters recorded (galvanic skin response, palm temperature, pulse rate, breathing rate). These modifications are in harmony with the physiological status of the women exposed to androstadienone. Furthermore, our results stress that although both of the ANS systems are antagonistically activated by androstadienone, the activation is strongly linked to the physiological hormone status, which results in agonistic effects on sexual and reproductive behaviour.

Here, we have focussed our evaluations on the effects of the physiological conditions of these women during (and after) their exposure to androstadienone by measuring their facial temperatures using high-resolution thermal infrared imaging. This technique demonstrates that exposure to androstadienone elicits different physiological responses that are mediated by activation of the ANS and that appear to result in both sympathetic-like and parasympathetic-like effects that are linked to hormone status. Androstadienone increased facial temperature in the ovulatory women, priming scattered patterns of temperature variations. Conversely, androstadienone reduced the superficial skin temperature of the face in the non-ovulatory women. This is in agreement with the physiological mechanisms of sexual arousal, which are reinforced when the sympathetic and parasympathetic systems are simultaneously activated to different degrees. In women, opposite hormonal asymmetries would induce opposite

sympatho-parasympathetic sexual asymmetries, such that general sensation and arousal are linked to the parasympathetic sexual system, while the local erogenous sensations are linked to the sympathetic system. In summary, the strong–weak balance of these two classes of hormones generates concomitant but asymmetric functioning of the sympatho-parasympathetic systems during sexual arousal (Motofei and Rowland, 2005). This autonomic balance can be elicited with hormones and/or modulated by pheromones, to reach the activation levels of each of the autonomic systems.

Furthermore, the asymmetric functioning of the sympatho-parasympathetic systems is highlighted by comparing these androstadienone effects with respect to the hormone status of the woman, as ovulatory versus non-ovulatory. Here, androstadienone appeared to cause a general thermal increase in the ovulatory women that was amplified in localised regions. These antagonistic effects resulted in regionalised oscillations of the temperatures in specific scattered patterns. Moreover, thermal fluctuations that suggest a double heat-peak response to androstadienone were detectable: an early response over the first few minutes of activation that is in agreement with previous studies (Jacob and McClintock, 2000), and a delayed response that is compatible with middle-to-late responses (Grosser et al., 2000; Bensafi et al., 2003).

Even though we did not specifically investigate late responses, the behaviour seen resembles a two-fold pheromone effect of androstadienone. In ovulating woman, androstadienone would act as a releaser pheromone, as represented by the early peak. Androstadienone can then operate as a modulator pheromone, as characterised by the late peak (McClintock, 2000). This dual action allows us to speculate on the activation of specific patterns of sexual and reproductive behaviour according to the ovulatory condition, at least in terms of sexual arousal. In contrast, with the non-ovulatory condition, only the modulator effect of androstadienone was preserved, as represented by the late peak. Early activation was also seen here, although, in contrast, it resulted in

a temperature decrease, which could represent a negative releaser effect to avoid sexual and reproductive behaviour.

These thermal fluctuations due to androstadienone exposure and their physiological effects across the menstrual cycle are congruent with variations in other autonomic parameters. As already suggested for olfactory perception, around ovulation, women recognize male odour as a pleasant smell, with the tendency thus to make contact with a potential partner. Conversely, in a non-ovulatory period (when there is less chance of pregnancy), the chemosignals that respond to the presence of a male can evoke unpleasant sensations in women (Watanabe et al, 2002). Moreover, these differences in autonomic responses are not linked to variations in olfactory thresholds. Pheromone perception appears to be constant across hormone status (Hummel et al, 1991), while the response to this stimulus is strikingly dependent on hormone status. An evolutionary hypothesis also comes from our thermal data, which is further supported by these results of autonomic responses to non-conscious perception of the male sexual pheromone: the pheromone can elicit specific neuroendocrine activation that regulates sexual and reproductive behaviour.

A new expanding scientific concept is that women have not lost oestrus (Gangestad and Thornhill, 2008). Our findings support the evidence that when in the fertile periods of their cycles, women are sexually attracted by physiological signals, such as pheromones. Through our ancestral evolution, pheromones were ‘designed’ for the assessment of predisposed displays of the genetic/ zoological quality of a mate, which we have shown still to be the case here. Thus dual sexuality of women has been hypothesised through the course of human evolution (Gangestad and Thornhill, 2008): the oestrus phase, which is related to the female sexual choice for good genes for their offspring, and the non-fertile phase, in which sexual and reproductive behaviour reduce male aggression towards offspring by uncertainties over paternity or as a form of

grooming for pair-bonding and couple reinforcing. These appear to be further supported by our findings.

Today, now 50 years after the discovery of pheromones (Karlson and Luscher, 1959), investigations into the influence of chemical communication on human sexual and reproductive behaviour are unveiling the evolution of its plesiomorphic nature.

Methods

Fourteen healthy women (mean age, 23 ±3 years) were enrolled in this study. The exclusion criteria were for an irregular hormonal cycle, use of contraceptive pills, smoking, alcohol or drug consumption, impaired sense of smell, homosexuality, any overt pathology or disease, or recent clinical surgery or anaesthesia. The women all provided written informed consent for participation in the study, which was performed in agreement with the ethical standards of the Helsinki Declaration, 1964, and approved by the local Human Board Review and Ethical Committee. Potential participants were a-priori tested for general olfactory perception and thresholds according to the Connecticut Chemosensory Clinical Research Centre test.

Androstadienone (Steraloids, Inc., Newport, RI) was used in its odourless volatile crystalline form (1 ×10-3 g/vial), to avoid problems linked to solvents and concentrations (Bensafi et al, 2004; Savic et al, 2001). The hidden vials that contained either placebo or androstadienone were changed for each woman for each session, with the contents of all of the vials later confirmed by gas chromatography. Subjects rested in comfortable supine position during the experiments, which were performed at the same time of the day in order to avoid possible circadian effects.

The data were analysed using paired t-tests within subjects (statistical significance threshold, $p < 0.05$), comparing conditions (ovulatory vs. non-ovulatory) and stimuli (androstadienone vs. placebo) through each experimental phase (adaptation, stimulation and recovery).

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Competing interests statement

The authors declare that they have no competing financial interests.

Author Contribution

A. M. and A. M. designed and performed experiments, analysed data and wrote the paper. G. L. R. and L. T. supervised the project.

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Figure 1. High-resolution infrared thermography facial images

The women underwent a total of four measurement sessions: two sessions (control and experimental) for each ovulatory condition (ovulatory, OV; non-ovulatory, NOV). Each woman was randomly assigned their measurement session sequences. Ovulatory status was determined according to urinary concentrations of luteinising hormone, using a commercial test kit (Clearplan, Schering spa). Each woman followed a series of preparatory standardisation procedures for thermal imaging (Merla, 2007) and a 20-min acclimatisation period in the climate-controlled measurement room (23 ±1 °C; 55%-60% relative humidity; no direct ventilation). The facial thermal imaging was performed using a 320×240 SC3000 QWIP Flir camera (NETD = 0.04 °C and 30 °C; sampling rate, 25 full frames/s). Representative thermal images are shown for placebo (Ctrl) and androstadienone (AND) exposure across the blinded experimental periods: 5-min adaptation phase (AP), with exposure to placebo (double-distilled water); 10-min stimulation phase (SP), with exposure to placebo or androstadienone (control and experimental, respectively); and 20-min recovery phase (RP), with exposed to placebo again. A false-colour temperature scheme is used for visualisation of the thermal effects (see inset).

Figure 2. Group-averaged normalised facial skin temperatures

The thermal image time series were re-aligned, corrected for movement artefacts, and processed according to experimental group using in-house-developed Matlab routines. The temperature curves in the control experiments showed linear drift for the women for both their ovulatory and non-ovulatory conditions. This trend appeared due to the resting supine position maintained

by the women during the recording sessions. These linear control trends were therefore removed from the temperature curves, with the normalised temperatures (TN) shown for the ovulatory and non-ovulatory women for placebo (Ctrl) and androstadienone (AND) exposure across the experimental adaptation (AP), stimulation (SP) and recovery (RP) periods. The ovulatory and non-ovulatory temperatures for the androstadienone exposure during the stimulation and recovery phases are significantly different from the placebo exposure ($p<0.05$, $p<0.001$, respectively; paired t-tests).

Figure 3. Pixel-by-pixel temperature subtraction

Representative pixel-by-pixel temperature subtractions of the thermal images for the ovulatory (OV) and non-ovulatory (NOV) women for placebo (Ctrl) and androstadienone (AND) exposure across the experimental adaptation (AP), stimulation (SP) and recovery (RP) periods. A false-colour temperature scheme is used for visualisation of the thermal effects (see inset).

Figure 4. Regional group-averaged normalised facial skin temperatures

After movement correction and image re-alignment, the temperatures of the ROI were adjusted for baseline and normalised (TN). Comparisons are shown for the ROI group thermal image series in the ovulatory and non-ovulatory women for placebo (Ctrl; for clarity, only non-ovulatory placebo is shown) and androstadienone (AND) exposure across the experimental adaptation (AP), stimulation (SP) and recovery (RP) periods. Facial regions: lachrymal, right (Lr), left (Ll); corrugator muscle, right (Cr), left (Cl); forehead (F), and nose tip (N).

With the ovulatory women, Lr and N show an early response peak with androstadienone and a delayed peak during recovery, the latter of which is also seen for the non-ovulatory women for Ll, Cl, F and N.

Figure 5. Group-averaged autonomic parameters

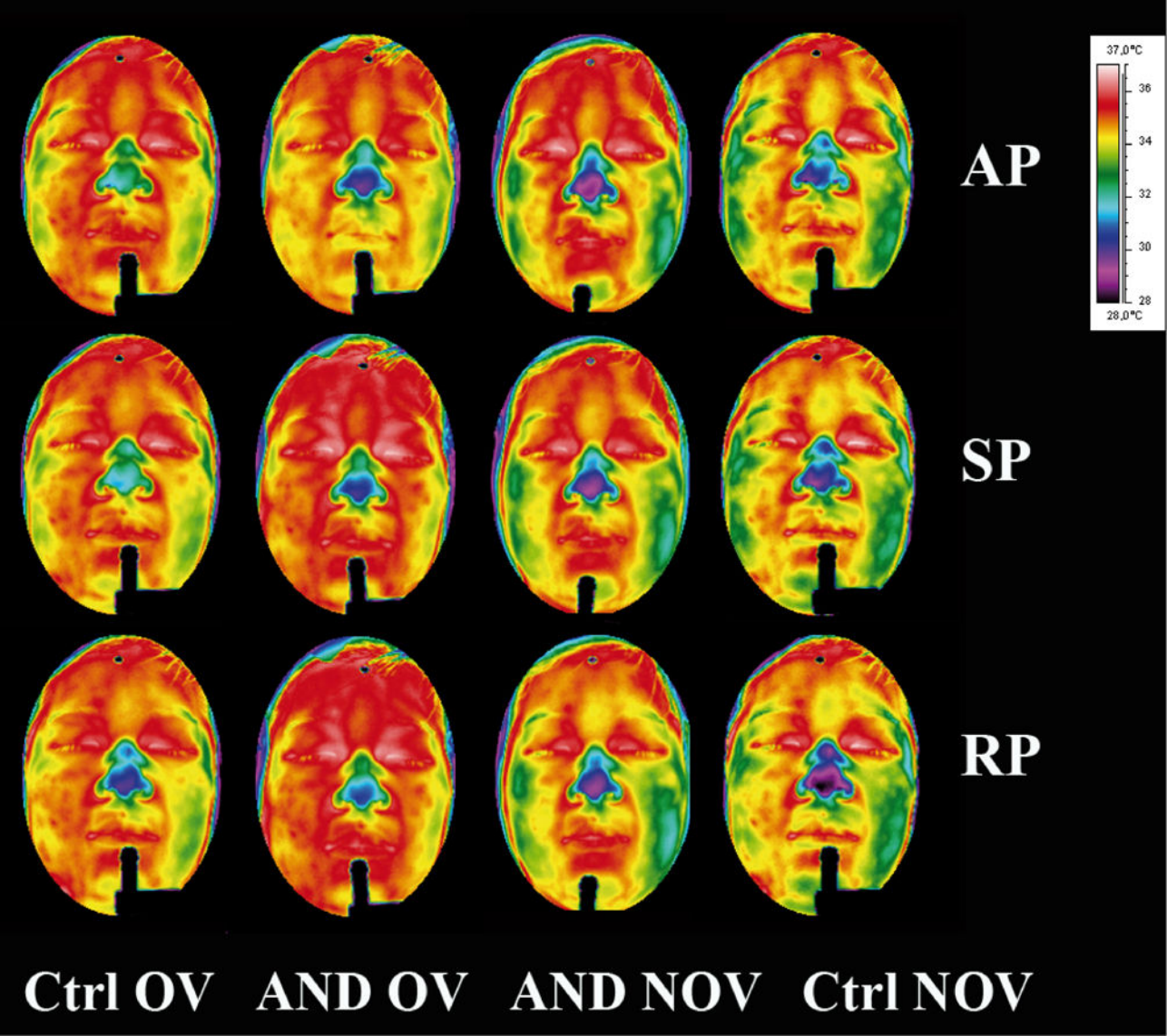
The autonomic parameters (pulse rate, breathing rate, galvanic skin response [GSR] and palm temperature) were recorded simultaneously with the facial thermal imaging (ADInstruments ML870 PowerLab 8/30). The GSR and palm temperature autonomic parameter curves were adjusted for baseline and normalised (μSN and TN, respectively), with pulse rates and breathing rates unadjusted. Comparisons are shown for these autonomic parameters in the ovulatory and non-ovulatory women for placebo (Ctrl; for clarity, only non-ovulatory placebo is shown) and androstadienone (AND) exposure across the experimental adaptation (AP), stimulation (SP) and recovery (RP) periods. In autonomic parameters result statistical differences when pheromone is applied (see table 1).

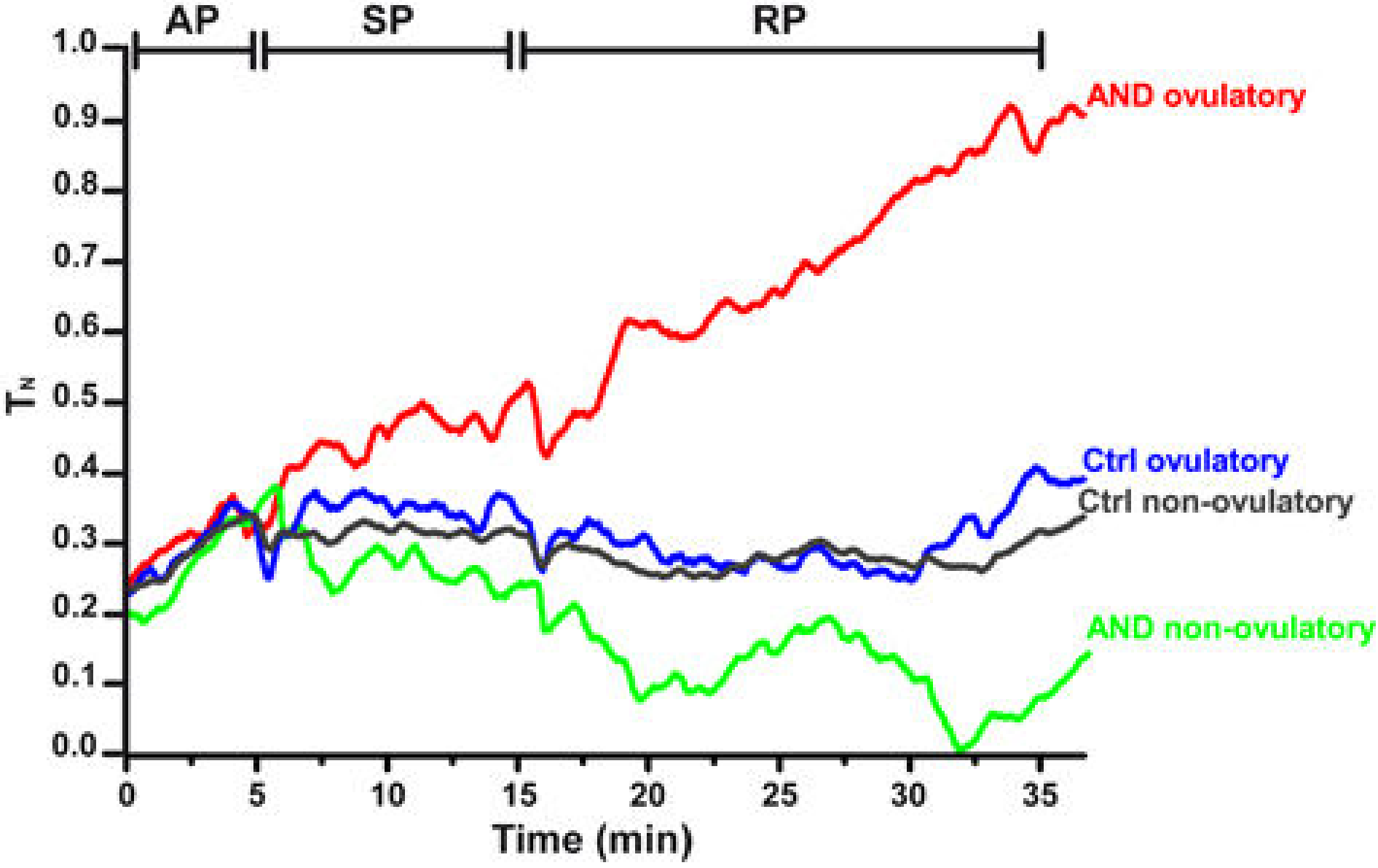
Table 1. Paired t-test results for the significance of conditions (ovulatory vs. non-ovulatory) and stimulus (androstadienone vs. placebo) across the experimental phases, and according to the recorded parameters.

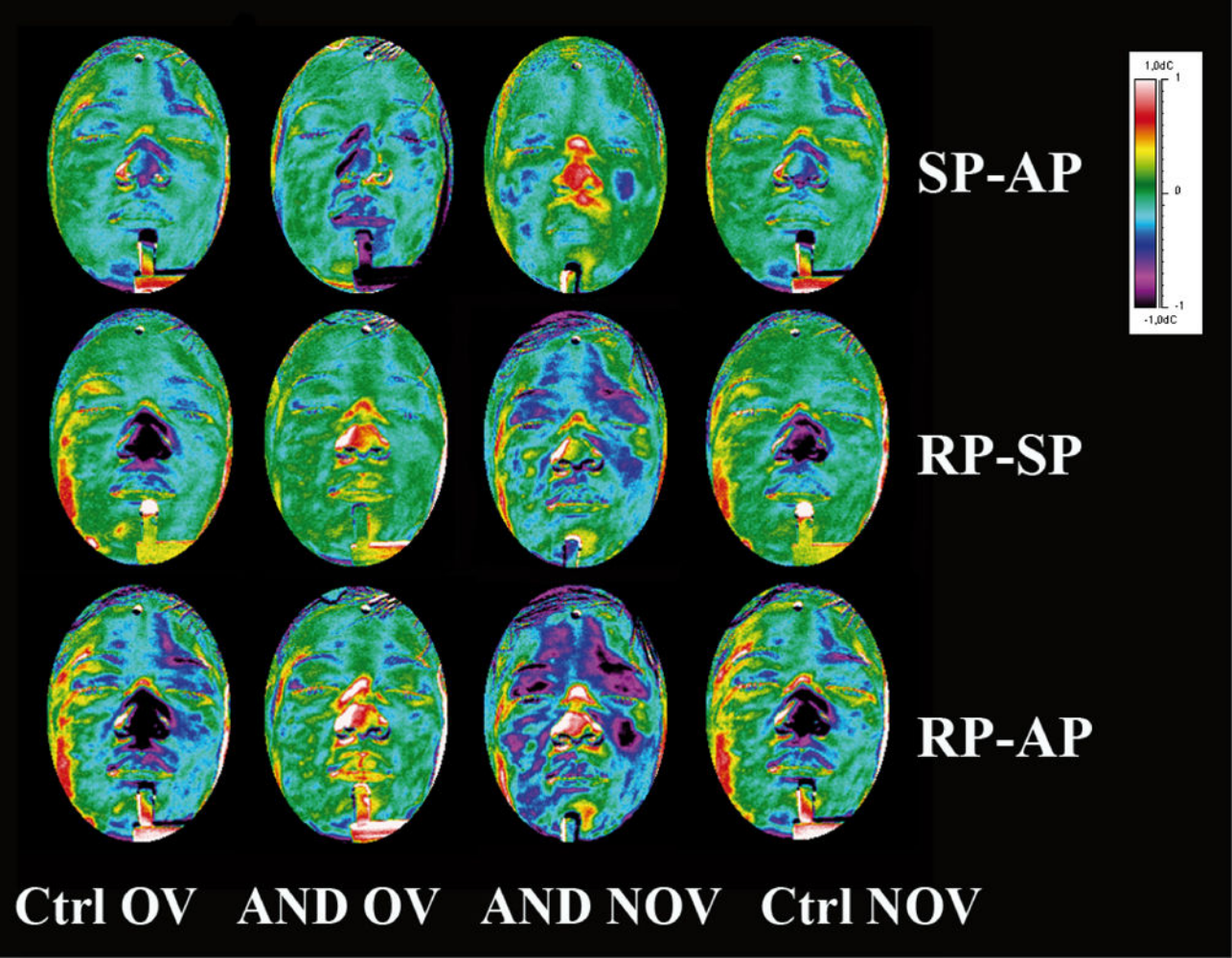
	Adaptation		**Stimulation**		**Recovery**	
Parameter	**ovulatory *versus* non-ovulatory**	**androstadienone *versus* placebo**	**ovulatory *versus* non-ovulatory**	**androstadienone *versus* placebo**	**ovulatory *versus* non-ovulatory**	**androstadienone *versus* placebo**
Lachrymal, right	ns	ns	**	**	**	**

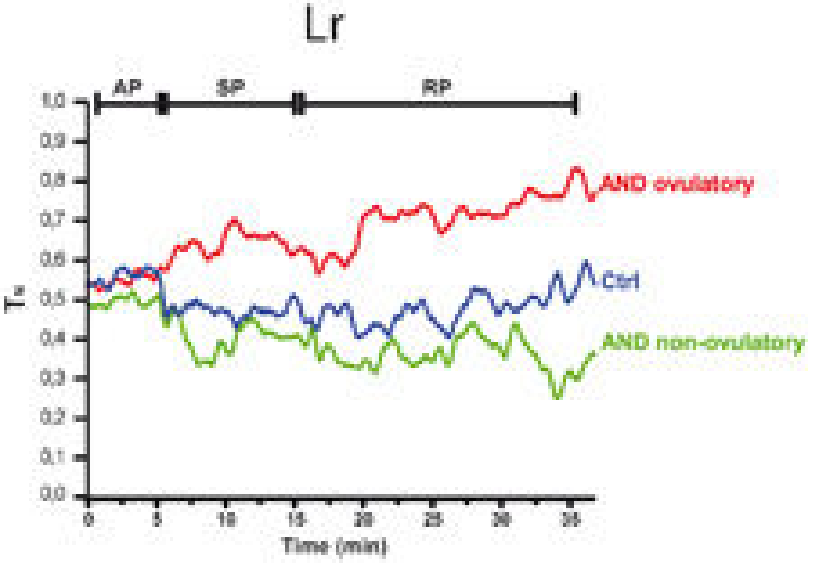
Lachrymal, left	ns	ns	**	**	**	**
Corrugator muscle, right	ns	ns	**	**	**	**
Corrugator muscle, left	ns	ns	**	ns	ns	**
Forehead	ns	ns	**	ns	**	**
Nose	ns	ns	*	*	**	**
Galvanic skin response	ns	ns	**	**	**	**
Palm temperature	ns	ns	**	**	**	**
Pulse rate	ns	ns	**	**	ns	**
Breathing rate	ns	ns	**	*	ns	**

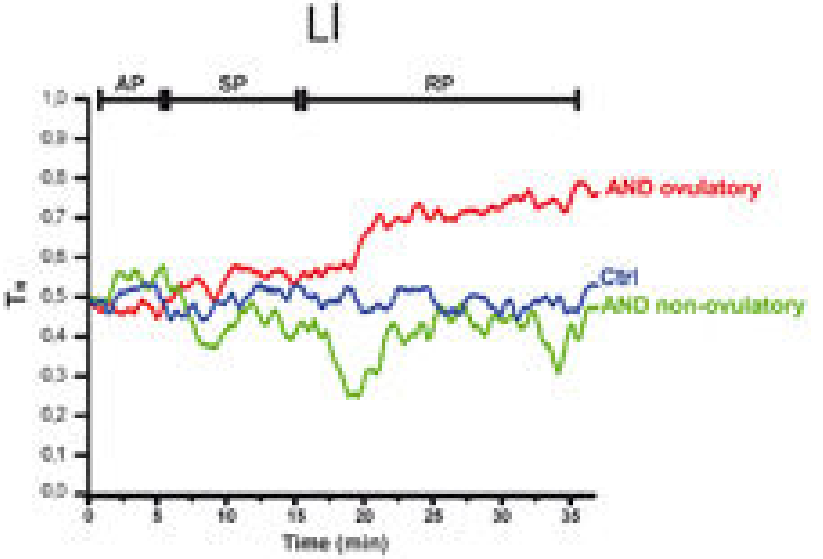
Placebo, double-distilled water; ns, not significant; *$p < 0.05$; **$p < 0.01$.


37,0°C
36
34
32
30
28
28,0°C
AP
SP
RP
Ctrl OV
AND OV
AND NOV
Ctrl NOV


AP
SP
RP
AND ovulatory
Ctrl ovulatory
Ctrl non-ovulatory
AND non-ovulatory
T_N
1.0
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
0
5
10
15
20
25
30
35
Time (min)


SP-AP
RP-SP
RP-AP
1,0dC
1
0
-1
-1,0dC
Ctrl OV
AND OV
AND NOV
Ctrl NOV


Lr
AP
SP
RP
AND ovulatory
Ctrl
AND non-ovulatory
Ts
Time (min)


Ll
AP
SP
RP
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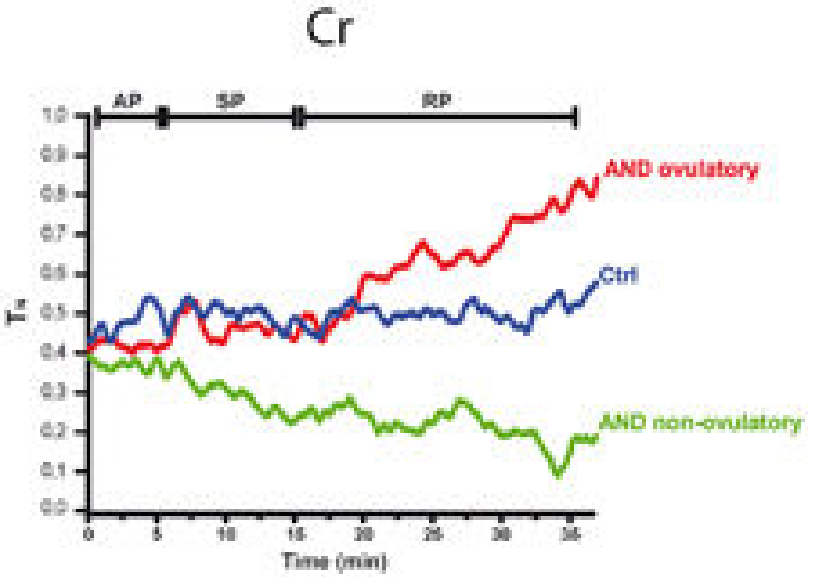
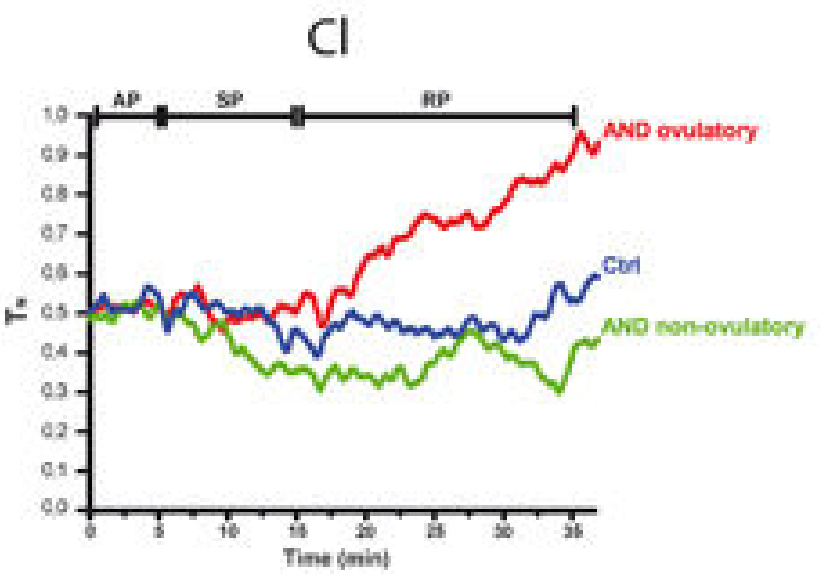
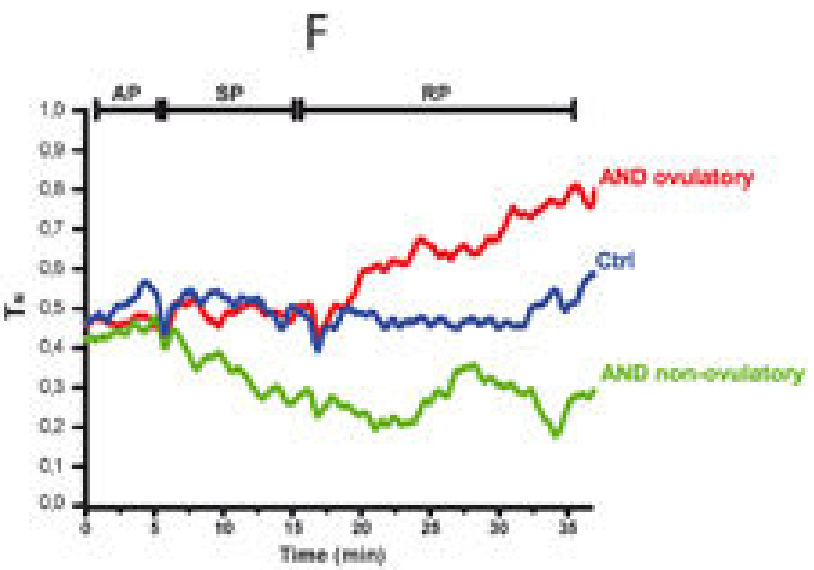
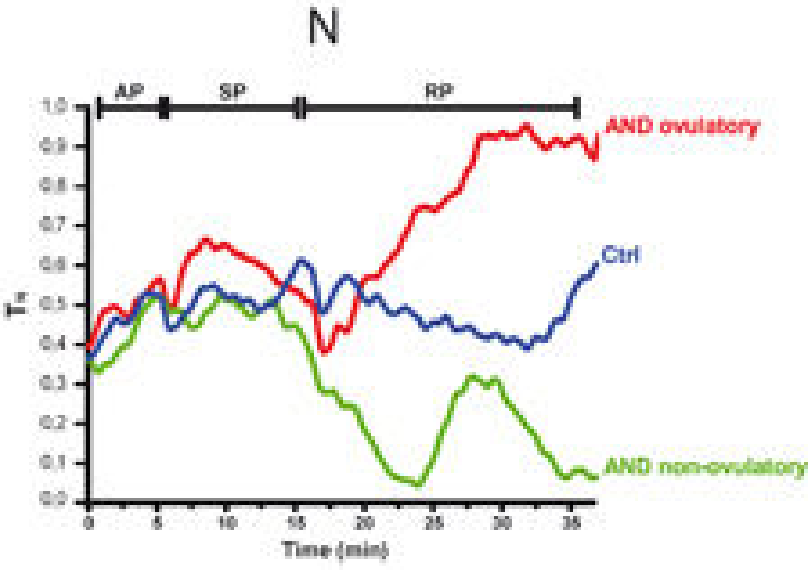
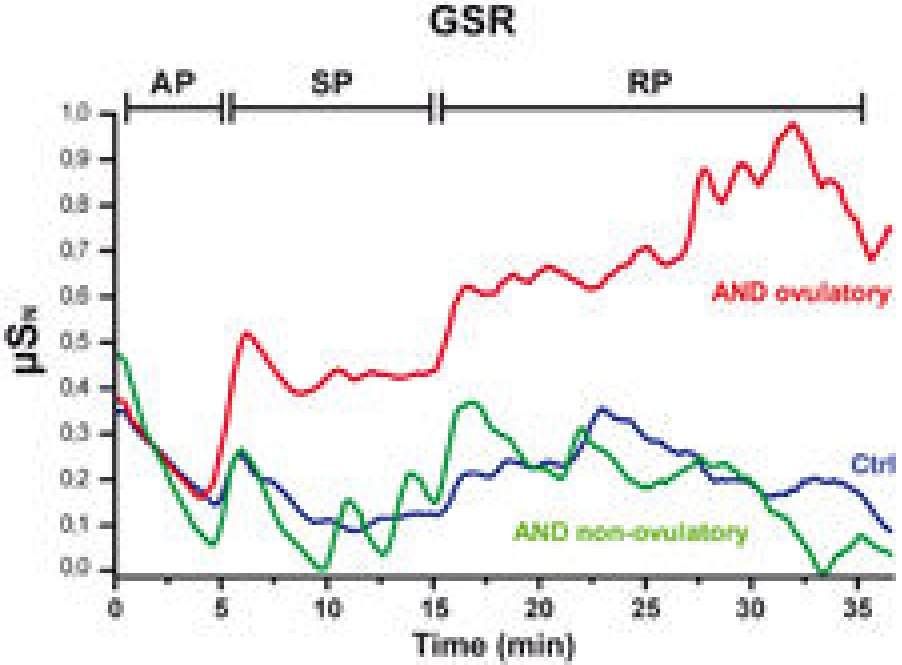
Ctrl
AND non-ovulatory
Ts
Time (min)

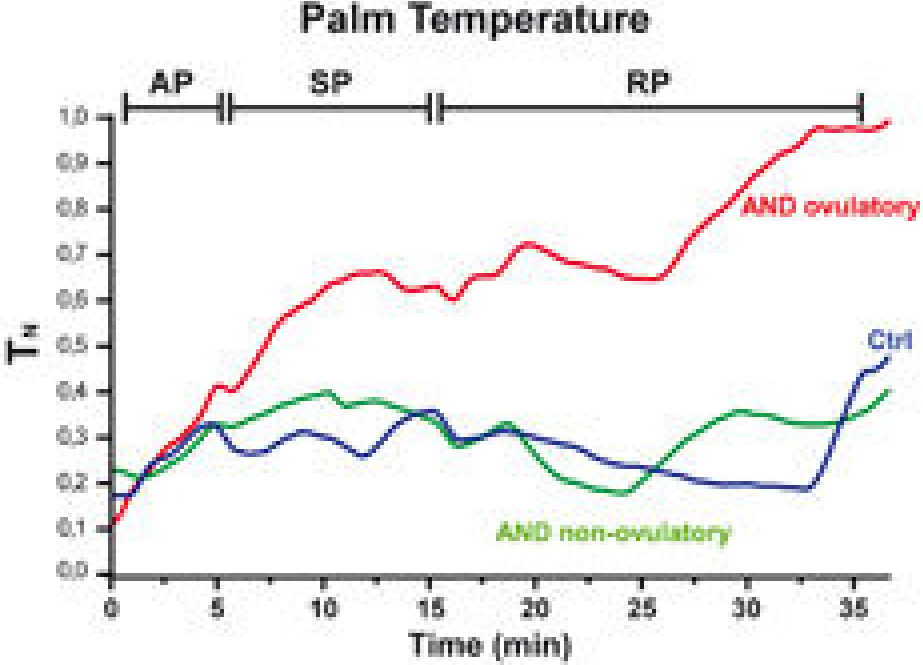

Cr
AP
SP
RP
AND ovulatory
Ctrl
AND non-ovulatory
Ts
Time (min)

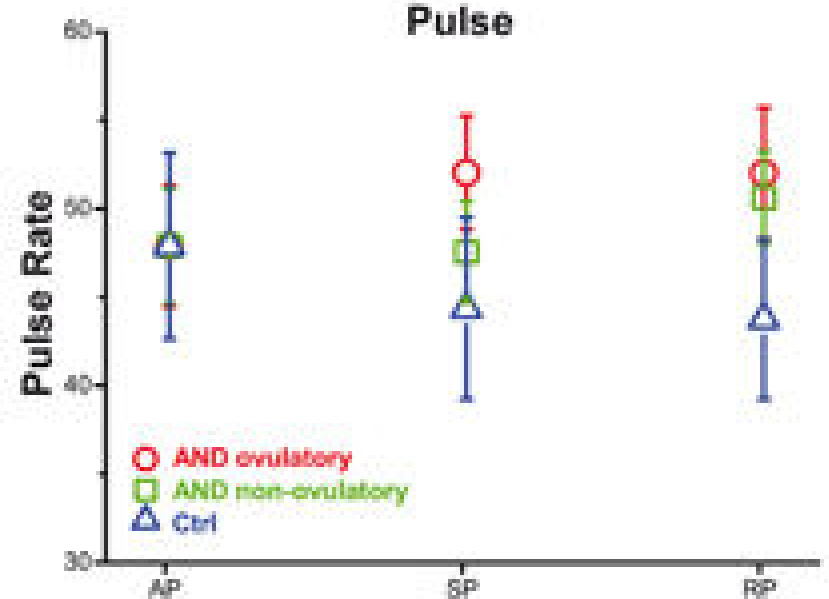

Cl
AP
SP
RP
AND ovulatory
Ctrl
AND non-ovulatory
Ts
Time (min)

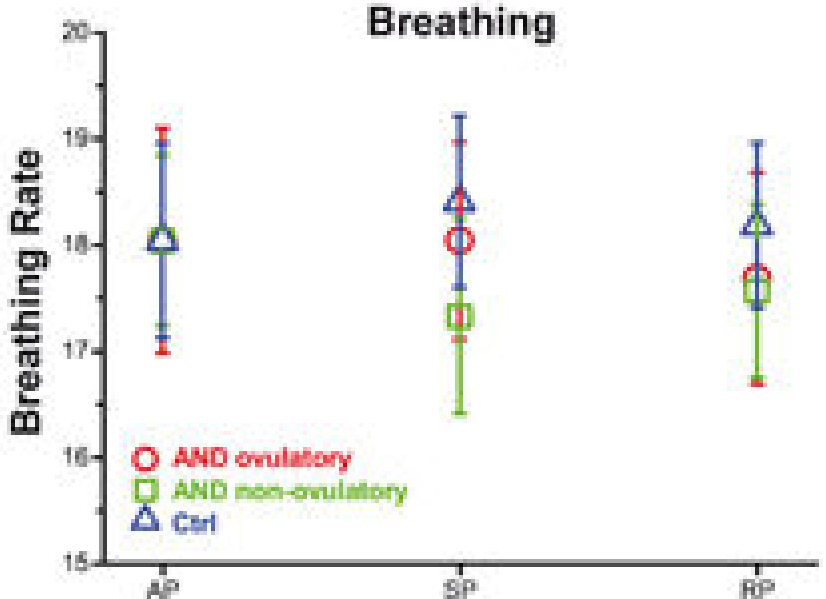

F
AP
SP
RP
AND ovulatory
Ctrl
AND non-ovulatory
Ts
Time (min)


N
AP
SP
RP
AND ovulatory
Ctrl
AND non-ovulatory
Ts
Time (min)


GSR
AP
SP
RP
μS
AND ovulatory
Ctrl
AND non-ovulatory
Time (min)


Palm Temperature
AP
SP
RP
T
AND ovulatory
Ctrl
AND non-ovulatory
Time (min)


Pulse
Pulse Rate
AND ovulatory
AND non-ovulatory
Ctrl
AP
SP
RP


Breathing
Breathing Rate
AND ovulatory
AND non-ovulatory
Ctrl
AP
SP
RP